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American Journal of Diseases of Children

VOLUME 35 JANUARY, 1928 NUMBER 1

CARDIAC FAILURE IN CHILDREN UNDER FIVE YEARS OF AGE

WITH SPECIAL REFERENCE TO ACUTE RHEUMATIC MYOCARDITIS *

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AND
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The occurrence in the hospital within a comparatively short space of time of several unusual examples of sudden myocardial failure in very young children has led us to make this communication. There seems little doubt that heart failure not due to congenital cardiac malformations or to diphtheria is a rare condition in children under 5 years of age. Whereas such cases have attracted attention chiefly from an etiologic standpoint, they likewise afford considerable interest both diagnostically and therapeutically.

Poynton [1] has emphasized the rarity of the first attacks of rheumatic carditis being fatal and has given brief histories of the cases of three children under 5 years of age observed in his clinic. Diagnosis did not offer any difficulties, as one or more of the usual extracardiac rheumatic manifestations were present. Death occurred within a few weeks after the onset of symptoms. He regards primary carditis with failure as one of the most fatal forms of rheumatic heart disease, and draws attention to the fact that it is more often encountered in young children.

In an article on the cutaneous manifestations of rheumatic fever in children, Bass [2] has mentioned a child of 3 years who had an attack of acute myocarditis followed by myocardial failure and death. There was a history of acute tonsillitis two weeks before the onset and one week later the appearance of erythema multiforme. Autopsy showed a fresh rheumatic endocarditis and great numbers of Aschoff bodies. The child had been seen one month before death, and the results of examination at that time were negative.

* Received for publication, Aug. 25, 1927.

* From the Department of Pediatrics of the Johns Hopkins University School of Medicine and the Harriet Lane Home of the Johns Hopkins Hospital.

1. Poynton, F. J.: Observations on the Nature and Symptoms of Cardiac Infection in Childhood, Brit. M. J. **1**:417, 1918.

2. Bass, M. H.: The Cutaneous Manifestations of Acute Rheumatic Fever in Childhood, M. Clin. N. Amer. **2**:201, 1918-1919.

Schroeder [3] has described a case of a white girl, aged 20 months, with a vague history of irritability of six weeks' duration. When first seen, she presented the typical manifestations of myocardial insufficiency. During the previous week it had been noted that her breathing was rapid. She developed a pericardial friction rub, but the etiology of the carditis remained uncertain until a few days later, when she exhibited typical choreiform movements. Death followed within a short time, and autopsy revealed small vegetations on the mitral valve, a nonpurulent pericarditis and Aschoff bodies in the myocardium.

Smith [4] has reported the case of a child, aged 2 years and 2 months, who after a brief illness died of myocardial failure. Postmortem examination showed some early vegetations on the mitral valve and Aschoff bodies throughout the heart muscle. Joint manifestations or subcutaneous fibroid nodules were not present. This author likewise emphasizes the rarity of rheumatic infection in children under 5 years of age, and the grave prognostic significance of a primary myocarditis with failure.

Three interesting cases of rheumatic myocarditis with failure and death in children under 2 years of age have been reported by Denzer.[5] His first case was strikingly similar to that reported by Smith. A white girl, aged 23 months, after an acute illness of five days' duration, died of cardiac failure, the rheumatic etiology of which was established by autopsy. There was no antecedent history of disease of the joints nor could any subcutaneous fibroid nodules be found. The other two cases, although similar, were associated with arthritis and subcutaneous fibroid nodules. Denzer suggests that many of the cases of "idiopathic myocarditis" described not infrequently in the past were rheumatic in origin.

REPORT OF CASES

CASE 1.—*History.*—O. S., a negro boy, aged 5 years, whose family history was unessential and whose birth and development were normal had had measles but none of the other diseases of childhood. A history of rheumatism could not be obtained. The present illness commenced just one week before admission, with slight abdominal pain and cervical rigidity. During the first two days he complained of vague pains in the knees and wrists. There was no swelling or redness of the joints, and the pains disappeared at the end of forty-eight hours. His condition apparently improved, and he was fairly comfortable the next few days, but on the night before admission his condition suddenly became serious. He vomited several times, his respirations became rapid, and he had occasional paroxysms of coughing. His temperature was 100 F. on admission to the hos-

3. Schroeder, L. C.: Observations on the Etiology and Pathology of Chorea Minor, J. A. M. A. **79**:181 (July 15) 1922.

4. Smith, A. de G.: Rheumatism and Its Manifestations in Children Under Five Years, Arch. Pediat. **39**:799, 1922.

5. Denzer, B. S.: Rheumatic Heart Disease in Children Under Two Years of Age, J. A. M. A. **82**:1243 (April 19) 1924.

pital, and he was extremely ill. The respiratory distress was so marked that only slight relief was obtained by placing the patient in the sitting position.

Examination.—Examination showed a poorly nourished boy about 10 pounds (4.5 Kg.) underweight. The tonsils were large and moderately infected, but an exudate could not be seen. The heart was slightly enlarged both to the left and to the right. The sounds were weak and the cardiac rate was rapid, but murmurs were not heard. There were a few fine moist râles at the left base. The liver was greatly enlarged and extended just below the level of the umbilicus. It was tender, and there was a questionable pulsation. There was no subcutaneous edema. Evidences of diphtheritic palsy were not present, and subcutaneous fibroid nodules were not found. It was evident that the child was suffering from acute myocardial failure. Death occurred one-half hour after admission.

Autopsy.—The heart was dilated and weighed 150 Gm. There was no gross evidence of pericarditis. The myocardium contained numerous Aschoff bodies. The lesions appeared fresh, and there was nothing to suggest a previous infection. The mitral valve was a little thickened, and at one point a small hyaline vegetation was seen. The left auricular wall and a few small areas of the pericardium were thickened and infiltrated with round cells. There was a pleural effusion on the left side amounting to about 100 cc. of clear serous fluid. The lungs showed a thickening of the alveolar walls with considerable congestion of the capillaries. The alveoli contained a small amount of fluid and some large phagocytic cells. The liver was enlarged, and although there was no actual chronic passive congestion, the capillaries around the central veins were greatly engorged with blood. The spleen was slightly enlarged, but all the other organs were normal.

Case 2.—B. L., a white boy, aged 2 years and 4 months, whose family history was unessential and whose birth and development were normal, had been seen in the outpatient department at the age of 7½ months with frank pneumonic consolidation of the right upper lobe. The recovery was uneventful. A history of rheumatism or any of the usual infectious diseases could not be obtained. The present illness began one week before admission, with dyspnea, vomiting, fever and marked restlessness. His condition became progressively worse. Later he coughed a great deal and became slightly cyanotic and drowsy.

On admission, his temperature was 99 F., his pulse rate 160 and respirations 54. The white blood cell count was 32,000 per centimeter, with 75 per cent polymorphonuclears. Examination showed a well developed boy, acutely ill and in marked respiratory distress. There was a slight generalized anasarca. The cyanosis was striking. The pharynx and tonsils were red, and there was a small amount of purulent postnasal discharge, but a membrane was not present. The heart was enlarged 4 cm. to the left of the mammary line. A definite point of maximal impulse could not be made out, and thrills could not be felt. A systolic shock was easily palpable in the epigastrium. A loud systolic murmur was heard over the precordium with maximal intensity in the sixth left interspace, where a soft diastolic murmur was also heard. The lungs were entirely clear. The liver was enlarged and extended to the level of the umbilicus. The spleen was just palpable. The child grew steadily worse, and died eleven hours after admission. Unfortunately, permission for autopsy could not be obtained.

Case 3.—S. S., a negro boy, aged 4 years, whose family history was unessential and whose birth and development were normal, had previously been admitted to the hospital at the age of 15 months with Vincent's angina, cervical adenitis and rickets. Physical examination at this time showed a normal heart. While

in the hospital he developed measles, recovered promptly, and was discharged a month and a half later in good condition. He remained entirely well until at the age of 4 years. A history of rheumatism or diphtheria could not be obtained. The present illness began three weeks before admission with an attack of generalized edema, which appeared suddenly and which was accompanied by the passage of what was said to be reddish-colored urine. Four days later, he had practically recovered, and he remained fairly well until three days before admission to the hospital. At this time, he developed marked dyspnea and slight edema of the feet.

Examination showed a fairly well nourished colored boy with marked respiratory distress. The temperature was 98.6 F. The heart was enlarged about 2.5 cm. to the right of the sternum and extended about the same distance to the left of the nipple line. The sounds were diminished in intensity. The rate was rapid and a definite protodiastolic gallop rhythm was present. Murmurs were not heard. There were râles at the bases of both lungs. The liver was greatly enlarged and tender. Examination of the urine showed a small amount of albumin but no blood. He did not have arthritis, and rheumatic nodules could not be felt. The Wassermann, Schick and intradermal tuberculin tests were negative. The white blood cell count was 7,620.

The child was evidently suffering from acute myocardial failure. Morphine was administered, and also 3 grains (0.195 Gm.) of the powdered leaves of digitalis every six hours until clinical signs of digitalization appeared. His condition improved rapidly, and he was then given 1½ grains (0.0974 Gm.) of digitalis twice daily. The enlargement of the heart was confirmed by roentgen-ray examination. Electrocardiograms both before and after the administration of digitalis showed nothing of significance except left ventricular preponderance. The P-R interval was not prolonged. A few weeks later, a faint short systolic murmur could be heard at the apex. He was discharged a month after the onset in good condition.

Case 4.—G. B., a white girl, aged 2 years, and 10 months, had always enjoyed good health except for an attack of measles and otitis media at the age of 2 years. Neither a history of "growing pains" nor of acute arthritis could be obtained. The present illness began two weeks before admission to the hospital with a mild rhinopharyngitis, cough and slight elevation in temperature. Her family physician said quite definitely that at this time there was no evidence of diphtheria. Two or three days after the onset, the feet and ankles became slightly edematous, and the eyelids somewhat puffy. Vomiting occurred three or four times a day, but during the course of the next week, the edema gradually disappeared, and on admission the child was not acutely ill. The following significant facts were recorded in the examination: The state of nutrition was excellent. Despite the fact that the skin appeared extremely pale, the hemoglobin content was 65 per cent (Sahli). There was only slight pitting edema of the lower extremities. Her respirations numbered 60 to the minute and did not cause any apparent distress The temperature was 98.2 F. The heart was not remarkable, and the rate only moderately increased. The sounds were of normal intensity; there were no murmurs, and the blood pressure was 86 mm. systolic and 58 mm. diastolic. The white blood cell count was 10,600 with 60 per cent polymorphonuclears. The urine contained a trace of albumin and on one occasion gave a slight positive reaction for blood. A chemical analysis of the blood did not show retention of nonprotein nitrogen. The tuberculin, Wassermann and Schick tests were negative.

It should be emphasized that the possibility of explaining the child's condition on the basis of heart failure was entirely overlooked at this time, and it was thought

that she was convalescing from a mild attack of acute nephritis. The urine cleared up rapidly, and the child was discharged a week later.

The child remained fairly well until one month after discharge, when there was a recurrence of her initial symptoms. She was brought into the outpatient department with the complaint of vomiting and rapid breathing. The temperature was 101 F. and the respiratory rate 80 a minute. There was questionable impairment of the percussion note at the right base, but abnormal cardiac observations were not noted, the pulse rate being 144 to the minute and the blood pressure 92 mm. systolic and 68 mm. diastolic. It was thought that the child was developing pneumonia. A roentgenogram showed a small area of infiltration at the right base and slight enlargement of the shadow of the heart. She was seen again in the outpatient department a few days later, and at this time the liver was definitely enlarged. One week after the onset of her second acute illness, she was readmitted to the hospital. She was extremely ill; respiratory distress was marked, the rate varying from 80 to 100 a minute. There was moderate cyanosis of the lips and the nailbeds despite her pallor. The pharynx was clear. The breath sounds were normal, and râles were not heard. The heart was slightly enlarged to the left. There was a slight diminution in the intensity of the sounds. Murmurs were not audible, but the cardiac action was rapid, and there were occasional extrasystoles. The pulse was of a poor quality. The presence of ascites was questionable. The liver was huge and extended almost to the crest of the ilium; it was exquisitely tender on slight pressure. It was obvious at this time that the child was suffering with acute failure of the heart, and drastic therapeutic measures were indicated. One hundred and fifty cubic centimeters of blood were removed from a vein in the arm, morphine was administered and the administration of digitalis started. She received the equivalent of 12 grains (0.78 Gm.) of digitalis within eighteen hours (body weight, 27 pounds [12.2 Kg.]). The following day the patient's condition was greatly improved. The respiratory and cardiac rates approached normal, the liver diminished markedly in size, and the peripheral edema disappeared. The extrasystoles had become so numerous, however, that digitalis was discontinued. Three days after admission, an electrocardiogram showed normal mechanism, P-R interval, 0.14 seconds and a low potential of the Q-R-S complexes in all leads. The urine contained a trace of albumin but no blood. The blood pressure was 86 mm. systolic and 48 mm. diastolic.

The child's condition remained excellent until nine days after admission, when the temperature rose to 102.5 F., the respiratory rate mounted to 60 a minute, and the liver increased slightly in size. Digitalis was again administered to the extent of 12 grains (0.78 Gm.) within twenty-four hours. The improvement was immediate and remarkable, and the response so definite that it seemed advisable to maintain digitalization by the administration of three fourths of a grain (0.0486 Gm.) of the powdered leaves by mouth twice daily. The clinical course was uneventful thereafter except for an occasional bout of unexplained fever.

COMMENT

The problem of establishing an etiologic basis for cardiac failure in infants and young children is frequently difficult. The question of rheumatic fever always arises, but in the absence of any of the accepted extracardiac manifestations, a definite diagnosis cannot be made. Cardiac failure in diphtheria offers little diagnostic difficulty. Early in the

disease, it is usually associated with faucial lesions, and later with paralyses. Less frequently a case of mild diphtheria is overlooked and is followed by myocardial failure unassociated with other complications. A Schick test, although not conclusive, may be helpful in establishing a diagnosis. Heart failure resulting from congenital cardiac anomalies is fairly easy to recognize, although the exact nature of the lesion cannot always be determined. In failure resulting from the so-called idiopathic hypertrophy of the heart, the condition is apt to be confusing unless the child has been observed over a moderately long interval. We have recently seen a case in point, namely, that of a colored infant, 11 months of age, with congenital syphilis, pneumonia of three weeks' duration and typical signs of myocardial failure appearing one week before death. Autopsy showed a large heart, with hypertrophy chiefly of the right side. Histologically, the heart muscle, pericardium and valves were normal, and we were forced to conclude that this was a case of idiopathic hypertrophy rather than syphilitic myocarditis or heart failure in any way related to the pneumonia. Although some of the cases of supposed idiopathic hypertrophy of the heart are undoubtedly due to congenital coarctation of the aorta, there still remain a few cases in which adequate explanation for the enlargement is not apparent.

To date there are no specific criteria, clinical or anatomic, which enable one to link acute myocardial failure with the acute infectious diseases, typhoid, influenza, scarlet fever and so forth. Recent investigations tend to show that the circulatory disturbances in these diseases and pneumonia result from a different mechanism, and present a clinical picture different from that in the cases we have described.

In any differential diagnosis it is well to keep in mind some of the more obscure causes of cardiac failure in the age group we are discussing. Malformations of the chest, particularly severe thoracic rickets, with the usual associated pulmonary conditions, are capable of producing definite pathologic changes in the heart. In the cases described by Park and Howland,[6] marked cardiac hypertrophy, most often of the right ventricles, was found, and although actual myocardial failure did not occur, we feel that such a possibility is so real that it must be always considered in order properly to direct therapy.

Myocardial failure occurring in beriberi is rarely seen in this country. In such cases the extracardiac manifestations of the disease are of distinct aid in diagnosis. An interesting series of these cases have been reported by Vitug.[7]

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6. Park, E. A., and Howland, J.: The Dangers to Life of Severe Involvement of the Thorax in Rickets, Bull. Johns Hopkins Hosp. **32**:1, 1921.

7. Vitug, W.: Some Clinical Aspects of Acute Cardiac Beriberi in Adults, J. Philippine Islands M. A. **6**:380, 1926.

White's [8] statistics show definitely that endocarditis under the age of 2 years is most frequently the result of septic infections. Both in his cases and in a few similar ones observed here, death has usually occurred before the classic symptoms of myocardial failure appear.

Although an absolute diagnosis could be made in only one case, in our series, O. S., one is impressed with the remarkable similarity in the clinical pictures presented. Only by a careful study of the subsequent clinical courses will it be possible to establish a definite diagnosis of rheumatism in the cases in which the patients survived.

As has been pointed out by Abt [9] and others, cardiac failure in young children presents some rather unusual features. Although the composite picture is similar to heart muscle failure in the adult, certain manifestations are particularly likely to predominate. Marked acceleration of the respiratory rate and rapid engorgement of the liver are not infrequently the only observations present, especially during the early stages; in fact, it is often on these symptoms alone that the diagnosis must be based, although the classic signs of decompensation may appear subsequently. Examination of the heart itself often reveals little information. Determination of cardiac enlargement is exceedingly difficult because of the extreme dyspnea. Arrhythmias are infrequent, and likewise one is invariably surprised by the paucity of auscultatory symptoms, particularly of murmurs.

The efficacy of digitalis in the relief of heart failure in childhood has long been seriously questioned. We feel that the drug is extremely valuable, and often a life-saving measure, and we advance two possible explanations for the poor results hitherto reported. In the first place, digitalis therapy has been employed in a great variety of conditions, often without any rational pharmacologic basis. One must differentiate sharply between true heart muscle failure and circulatory collapse. During the past few years, work on the minute output of the heart, both on experimental animals and man, has broadened our understanding of the mechanism of cardiac failure. This work has recently been reviewed by Harrison and Leonard.[10] These authors, working on normal dogs, demonstrated a diminution in the cardiac output following the use of digitalis. Prior to this, Eppinger and his associates [11] had made the

8. White, P. D.: The Incidence of Endocarditis in Earliest Childhood, Am. J. Dis. Child. **32**:536 (Oct.) 1926.

9. Abt, I. A.: The Decompensating Heart in Childhood, Illinois M. J. **50**: 135, 1926.

10. Harrison, T. R., and Leonard, B. W.: The Effect of Digitalis on the Cardiac Output of Dogs and Its Bearing on the Action of the Drug in Heart Disease, J. Clin. Investigation **3**:1, 1926.

11. Eppinger, Hans; von Papp, L., and Schwarz, H.: Ueber das Asthma Cardiale, Berlin, 1924, p. 191.

surprising observation that heart failure is associated with increased cardiac output. With this last observation in mind, together with the conception that ventricular imbalance is present in heart failure, it seemed that a correction of these factors offered an explanation for the efficacy of the drug. They added that in the conditions in which the clinical picture indicated acute circulatory failure or shock, without evidence of visceral or peripheral congestion, digitalis is probably contraindicated. In fact, they go so far as to say that digitalis may possibly have a harmful action when the cardiac output is believed to be materially decreased and simultaneous evidence for lack of ventricular balance does not exist. In view of the foregoing facts, we feel that an accurate clinical analysis in each case is essential for the proper administration of the drug.

Furthermore, attention has recently been called to the fact that large doses of digitalis are necessary in order to secure satisfactory effects in children. Jacobsen and Davison [12] have recently published a series of cases demonstrating that digitalis is a distinct aid in heart failure of rheumatic origin in childhood. In proportion to body weight, most of their patients required about two and one-half times the adult dosage. Our patients required correspondingly large amounts to effect complete digitalization. We have just had the opportunity of observing the effect of digitalis on two cases of heart failure in infants. The histories of both children were almost identical. One was 14 months of age and the other 16 months. There was no evidence pointing to either a rheumatic or diphtheritic etiology. Their illnesses were of short duration, and on admission they were suffering from myocardial decompensation. They differed from the foregoing cases only in that their hearts were tremendously enlarged and the roentgenograms were similar to those in cases of idiopathic hypertrophy of the heart. Digitalis was administered, and was followed by prompt improvement. Although the etiology of the myocardial failure is still a matter of speculation, the response to digitalis was sufficiently conclusive to warrant the use of the drug in similar cases.

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12. Jacobsen, A. W., and Davison, W. C.: Digitalis Therapy in Cardiac Decompensation in Children, Am. J. Dis. Child. **32**:373 (Sept.) 1926.

EFFECT OF ULTRAVIOLET LIGHT ON THE BLOOD OF NEW-BORN INFANTS

II. ERYTHROCYTES AND HEMOGLOBIN *

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With the increasing use of ultraviolet light as an important therapeutic measure for children, it is obviously necessary to ascertain the effect of this procedure on the young organism. In the first paper of this series, it was shown that the ultraviolet light was capable of increasing the blood platelets in the new-born.[1] This paper deals with the effect on the erythrocytes and hemoglobin.

MATERIAL

This study is based on the observations on a series of 200 new-born infants, during a period of eight months. The erythrocyte count and hemoglobin determinations were made within six hours after birth, and at twenty-four hour intervals thereafter. The determinations were made at the same time every day. Treatments with the ultraviolet ray were begun on alternate infants on the second day, and continued thereafter at the same time on each of the following four days.

METHOD

The estimation of the hemoglobin content is unsatisfactory because of the lack of a uniform standard for all methods. Haldane[2] has considered the oxygen capacity of the blood of normal persons to be 18.5 cc., corresponding to 13.8 Gm. of hemoglobin in 100 cc. If the grams of hemoglobin in the blood of normal persons are determined by the oxygen capacity method, and expressed in percentage, considering 13.8 Gm., or 100 per cent, the resulting percentage of hemoglobin will be about 115 per cent, which is misleading. Haden,[3] using the Van Slyke oxygen capacity method, found the normal standard to be 15.6 Gm. of hemoglobin per hundred cubic centimeters.

As it is rather difficult to take from a new-born infant each day a quantity of blood sufficient for a determination of the oxygen capacity, the method of Newcomer[4] was used. As Sackett[5] has pointed out, the difficulty is to arrive at a

* Received for publication, Sept. 26, 1927.

* From the Department of Pediatrics, Rush Medical College of the University of Chicago, and the Presbyterian Hospital.

1. Sanford, H. N.: The Effect of the Ultraviolet Light on the Blood of New-Born Infants: I, Am. J. Dis. Child. **33**:50 (Jan.) 1927.

2. Haldane, J. S.: The Colorimetric Determination of Hemoglobin, J. Physiol. **26**:497 (June) 1900-1901.

3. Haden, R. L.: Normal Hemoglobin Standard, J. A. M. A. **79**:1496 (Oct. 28) 1922.

4. Newcomer, H. S.: A New Optical Instrument for the Determination of Hemoglobin, J. Biol. Chem. **55**:569 (April) 1923.

5. Sackett, G. E.: Hemoglobin and Iron in the Blood, J. Lab. & Clin. Med. **10**:1018 (Sept.) 1925.

suitable value for the average amount of hemoglobin, with a high erythrocyte count. His standard of 16.4 Gm. of hemoglobin to 100 cc. of blood with an erythrocyte count of 5,000,000 was found to be as close as ordinary technical errors would allow. The readings were checked weekly by several iron determinations of Wong[6] to obviate any changes in the colored glass standard.

In drawing the blood to make determinations of both hemoglobin and erythrocytes, the skin of the heel was thoroughly cleansed with green soap and water, and finally with alcohol. A deep puncture was then made on the heel, which resulted in an adequate flow of blood. To insure a free flow, three drops were allowed to fall as a routine; then the blood for the determination of hemoglobin was drawn, and that for the cell count was taken last. The cell count was made immediately, while the solution for the hemoglobin determination was set aside and read after twenty-four hours.

Treatments with the ultraviolet ray were given by means of an air cooled mercury vapor quartz lamps, of 110 volts, 60 cycle and 10 amperes. A voltage of 70 was always administered, and maintained throughout the exposure. The technic of

TABLE 1.—*Hemoglobin Content in Series of New-Born Infants*

	Normal						Ultraviolet Exposure					
	Maximum		Minimum		Average		Average		Minimum		Maximum	
Day	Gm. per 100 Cc.	Per Cent	Gm. per 100 Cc.	Per Cent	Gm. per 100 Cc.	Per Cent	Per Cent	Gm. per 100 Cc.	Per Cent	Gm. per 100 Cc.	Per Cent	Gm. per 100 Cc.
1	23.0	140	14.4	88	18.8	115	114	18.7	85	13.9	140	23.0
2	21.3	130	16.2	98	18.05	110	111	18.07	94	15.4	135	22.2
3	22.2	135	13.4	82	17.2	105	110	18.05	85	13.9	140	23.0
4	20.6	125	13.4	82	17.2	105	108	17.7	85	13.9	135	22.2
5	19.7	120	12.0	73	17.2	105	105	17.2	80	13.1	125	20.6
6	20.6	125	12.5	76	16.4	100	112	18.4	90	14.7	135	22.2
7	20.6	125	13.1	80	16.4	100	106	18.2	80	13.1	130	21.3
8	19.7	120	12.3	75	15.6	95	100	17.4	78	12.8	125	20.6
9	19.7	120	13.4	82	15.6	95	97	15.9	80	13.1	120	19.7
10	19.7	120	10.8	66	15.4	94	95	15.6	75	12.3	120	19.7

the exposures has been described in an earlier paper; the dorsal and ventral surfaces were first exposed for one minute each, and the time was increased one minute a day until the fourth day.

COMMENT

In the normal series, the number of erythrocytes compares favorably with that found by other investigators.[7] The variations between the count in sexes was within the limits of technical error, and not worth recording.

The results of the determinations of the hemoglobin content are of interest. Other investigators[8] have reported extremely high values

6. Wong, S. Y.: Colorimetric Determination of Iron and Hemoglobin in the Blood, J. Biol. Chem. **55**:421 (March) 1923.

7. Lucas, W. B.; Dearing, B. F.; Hoobler, H. R.; Cox, A.; Jones, M. R., and Smyth, F. S.: Blood Studies in the New-Born, Am. J. Dis. Child. **22**:525 (Dec.) 1921. Lippman, H. S.: A Morphologic and Quantitative Study of the Blood Corpuscles of the New-Born, Am. J. Dis. Child. **27**:473 (May) 1924.

8. Williamson, C. S.: Influence of Age and Sex on Hemoglobin, Arch. Int. Med. **18**:505 (Oct.) 1916. Appleton, V. B.: Determination of Hemoglobin During Infancy by the Palmer and Van Slyke Methods, J. Biol. Chem. **34**:369 (May) 1918.

during the first week of life. It will be noted that while the maximum value compares with these high results, the minimum is much lower. The average is considerably lower than is generally supposed, although similar results have been reported by Lucas and others.[9] Two patients in the series were considered to be in a pathologic condition, and, therefore, the cases were not tabulated. One had a hemoglobin content of 40 per cent and 3,000,000 red cells; the other, a hemoglobin content of 45 per cent and 3,800,000 red cells. The physical observations in these infants were normal. Anemia in the new-born is considered rare, only five cases[10] being reported in the literature. My results in these two cases would indicate that anemia is not uncommon but is not diagnosed because a blood count is not made. Both infants gained normally, and examinations of the blood made eight weeks thereafter revealed: a hemoglobin content of 75 per cent, and 4,200,000 erythrocytes, and a hemoglobin

TABLE 2.—*Erythrocyte Content in Series of New-Born Infants*

	Normal			Ultraviolet Exposure		
Day	Maximum	Minimum	Average	Average	Minimum	Maximum
1	8,000,000	4,200,000	5,800,000	5,800,000	3,000,000	8,000,000
2	6,400,000	4,200,000	5,500,000	5,700,000	3,200,000	7,600,000
3	7,600,000	4,000,000	5,200,000	6,000,000	4,600,000	8,000,000
4	7,000,000	4,800,000	5,000,000	5,900,000	5,000,000	7,600,000
5	6,800,000	5,000,000	5,000,000	5,500,000	4,500,000	7,000,000
6	6,500,000	5,000,000	5,100,000	6,000,000	4,200,000	8,000,000
7	6,500,000	4,400,000	5,200,000	6,000,000	4,000,000	6,800,000
8	6,000,000	4,800,000	5,000,000	5,500,000	4,500,000	6,500,000
9	6,000,000	4,200,000	4,800,000	5,000,000	4,000,000	6,000,000
10	6,000,000	4,000,000	4,500,000	4,800,000	4,000,000	6,000,000

content of 78 per cent and 4,400,000 erythrocytes, respectively. Both infants received treatments with the ultraviolet ray.

In the series of infants receiving treatment with the ultraviolet ray, there was a slight increase both in the number of red cells and in the hemoglobin content twenty-four hours after the first treatment. These results remained stationary for the next three days, and were slightly increased after the fourth day, following the last treatment. It must be emphasized that the results are a general average. Many cases did not show any increase, and a large number showed a decrease both in the number of red cells and in the hemoglobin content after treatment. It was necessary to treat a large number of patients to ascertain whether the general effect was one of an increased hemoglobin content and an increased number of erythrocytes.

9. Gundoben, N. P.: Die Besonderheiten des Kindesalters, Berlin, S. Rubinstein, 1912.

10. Bonar, B. E.: Anemia in the New-Born, Am. J. Dis. Child. **33**:226 (Feb.) 1927.

Some of the results obtained were spectacular, and in one case a hemoglobin content of 85 per cent and 4,500,000 erythrocytes were increased to a hemoglobin content of 115 per cent and 8,000,000 cells after four treatments with the ultraviolet ray. Occasional bizarre increases were also observed in normal cases. The two infants with anemia responded well to ultraviolet irradiation. The hemoglobin content rose 20 per cent in one case and 15 per cent in the other, after four treatments. The number of erythrocytes was increased by 1,000,000 in each case.

It was impossible to follow many of these cases after the patients were discharged from the hospital. The few that had repeated examinations of the blood showed normal values. From these counts and the general downward trend of the results after treatment, it may be assumed that the hemoglobin content and the red cell count drop rapidly to a normal level.

These values may explain the different results obtained by investigators in the effect of light and darkness on the number of erythrocytes and hemoglobin content. It has been a popular belief that workers in mines or in darkness suffered from a decrease in hemoglobin content and number of erythrocytes. Blessing,[11] a surgeon on the Nansen expedition, did not find any change in hemoglobin content or in the number of red cells in his fellow members after their exposure to the long polar nights. Grober and Sempell [12] examined horses kept for from five to ten years in mines and did not find a decrease in the hemoglobin content and the number of red cells. As to the effect of sunlight on the hemoglobin content and number of red cells, Graffenburger,[13] Marti,[14] Borisson,[15] Orum,[16] Aschenheim,[17] Koster [18] and Laurens and Sooy [19] all found a decrease in darkness and an increase in sunlight.

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11. Blessing, H. G.: On the Norwegian Fram Expedition, Deutsche med. Wchnschr. **23**:251 (April 15) 1897.

12. Grober, D., and Sempell, O.: The Blood Cells Under Long Deprivation of Sunlight, Deutsche Arch. f. klin. Med. **129**:305 (July) 1919.

13. Graffenburger, L.: Researches on the Absence of Light, Arch. f. d. ges. Physiol. **4**:253 (May) 1893.

14. Marti, A.: Effect of Darkness and Light on Red Blood Cells, Verhandl. d. Cong. f. inn. Med. **15**:587 (May) 1897.

15. Borisson, P.: Heliotherapy and the Blood, Ztschr. f. diätet. u. physik. Therap. **5**:337 (April) 1900.

16. Orum, H. P.: The Influence of Light on the Blood, Arch. f. Physiol. **114**:1 (Aug.) 1906.

17. Aschenheim, E., and Meyer, S.: The Influence of Light on the Blood, Ztschr. f. exper. Path. u. Therap. **22**:22 (Jan.) 1921.

18. Koster, K.: Blood Picture During Heliotherapy, Deutsche Ztschr. f. Chir. **160**:352 (Dec.) 1920.

19. Laurens, H., and Sooy, J. W.: Effect of Light and Darkness on the Blood, Proc. Soc. Exper. Biol. & Med. **22**:114, 1924.

Considering that the effect of sunlight is probably due to ultraviolet radiation, results have varied as to the effect of ultraviolet radiation on the blood. Aschenheim,[20] Gelera [21] and Barenberg [22] did not find an increase in hemoglobin and red cells, while Miles and Laurens [23] did.

In my series, a large number of infants did not show any change and a considerable number showed a definite decrease in the number of blood cells and the hemoglobin content. The most spectacular results were obtained in those patients who had a low hemoglobin content and a comparatively low cell count. Hobart [24] has found that blood regenerates much faster in light than in darkness and still more rapidly under ultraviolet radiation. Tixier [25] has emphasized, however, that ultraviolet irradiation is effective only in secondary anemia in blood regeneration.

All these results would indicate a stimulation of the hematopoietic organs. The increase in hemoglobin content during irradiation might be due to the decreased volume of blood resulting from loss of water from the blood during the first days of life. As there is an increase in the number of red cells in cases in which the hemoglobin is increased, however, it does not appear that there is an actual increase of the hemoglobin content in individual cells. The subject of blood volume in the new-born is not well understood and deserves more study.

SUMMARY

1. The percentage of hemoglobin in the blood of new-born infants is not so high as is usually quoted.

2. Short exposure to the ultraviolet light increases the hemoglobin content and number of red cells to a slight extent in an average number of cases; there is a rapid return to normal.

3. Short exposures to the ultraviolet light increase the hemoglobin content and number of red cells to a greater extent in cases in which the hemoglobin content and number of red cells are lower than normal; they tend to remain nearly normal.

30 North Michigan Avenue.

20. Aschenheim, E.: The Influence of Sunlight on Blood Cells, Ztschr. f. Kinderh. **9**:87 (Sept.) 1913.

21. Gelera, M.: Influence of Artificial Light on Blood Corpuscles, Gaz. d. osp. **43**:394 (April 23) 1922.

22. Barenberg, L. H.; Friedman, J., and Green, D.: The Effect of Ultraviolet Irradiation on the Health of a Group of Infants, J. A. M. A. **87**:1114 (Oct. 2) 1926.

23. Miles, A. L., and Laurens, H.: The Physiological Action of Darkness, Daylight and Carbon Arc Radiation: III, Am. J. Physiol. **75**:443 (Jan.) 1925.

24. Hobart, H.: Action of Light on Blood Regeneration, Klin. Wchnschr. **2**:1213 (June 25) 1923.

25. Tixier, R.: Ultraviolet Rays in Treatment of Anemia, Paris med. **57**:529 (Dec. 19) 1925.

SCARLET FEVER ANTITOXIN

REPORT OF FOUR HUNDRED AND SIXTY-FIVE CASES*

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It is evident from the numerous inquiries that are received at the Willard Parker Hospital that the true value of scarlet fever antitoxin is not generally known; with this in view, the following observations are presented.

The series presented consists of 465 consecutive and unselected cases, in which 250 of the patients did not receive antitoxin. I feel, therefore, that the real value of the antitoxin can be obtained.

All patients with scarlet fever who were admitted to the hospital did not receive antitoxin, for it was apparent that in the moderately severe and in the mild cases the patients did not show sufficient toxemia to warrant the treatment. Furthermore, the serum rash that may appear often causes more subjective symptoms than the disease per se. Therefore, it was decided that the temperature and the clinical observations should govern the administration of the antitoxin. As a rule, patients who had a temperature below 101.4 F. with little if any toxicity, and who presented few clinical signs and symptoms, did not receive the treatment.

The antitoxin that is used at this institution is prepared by the New York Health Department at Otisville, N. Y. At first a preparation that contained only antitoxic properties was used; later, at the suggestion of Dr. Park, a preparation that contained both antitoxin and antibactericidal properties was used, and it was found that this gave the best results.

In chart 1 the cases are grouped according to temperature, and show the effect observed when antitoxin is not given. It will be noticed that these are moderate cases.

The cases in chart 2 are grouped as those in chart 1 and illustrate the effects observed following antitoxin.

Table 1 illustrates the results obtained following intravenous and intramuscular therapy combined, and intravenous alone; the cases are representative.

In table 2, a collection of cases is presented in which no results were noted following the use of antitoxin.

It is now known that the degree of toxemia in scarlet fever varies a great deal, and in table 2, case 337 illustrates this fact. In this case

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a small dose of antitoxin was initially given to the patient; at the end of forty-eight hours, no improvement was noted. A second dose was given, and at the end of the following twenty-four hours the temperature had fallen to 99.2 F., where it remained; from this one may reasonably conclude that the delayed improvement was due to insufficient antitoxin. The results in cases 1270 and 952 suggest the possibility that several types of streptococci may cause scarlet fever, for in these cases, although a second dose of antitoxin was given, there was no change either in the temperature or in the clinical picture. Furthermore, cases are seen in which the patient is well advanced in the convalescent period when suddenly a picture of scarlet fever is presented.

It is difficult to state the exact influence of the antitoxin on the number of complications. This can readily be seen when one considers that only moderately ill and acutely ill patients receive the treatment, and

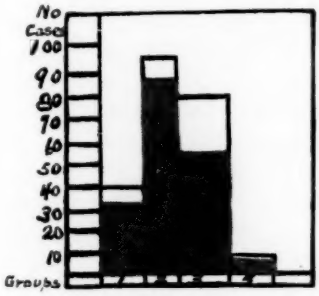

Chart 1

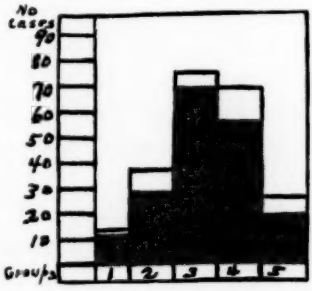

Chart 2

Chart 1.—Group 1 includes patients with a temperature of from 99 to 99.8 F.; group 2, from 100 to 100.8; group 3, from 101 to 101.8, and group 4 from 102 to 102.8. In this and in chart 2 the height of the column represents the number of cases; the shaded areas, the number of patients that had a temperature of 100 or below at the end of forty-eight hours.

Chart 2.—Group 1 includes the patients with a temperature of from 99 to 100.8; group 2, from 101 to 101.8; group 3, from 102 to 102.8; group 4, from 103 to 103.8 and group 5, from 104 to 105.

that the greatest number of complications occur in these cases. As a result, there is no group with which the cases may be compared. Observations, however, show that the number of complications which occur in this group are decreased. This may be illustrated by the fact that the total number of true scarlatinal complications after admission in the foregoing group was only 0.32 per cent higher than in the patients with lighter attacks who did not receive antitoxin.

There were five deaths in the 465 cases: one from empyema, three from pneumonia and one from peritonitis following a ruptured appendix.

CONCLUSIONS

1. Scarlet fever antitoxin is of definite value. All patients, however, should not receive this treatment, for, as is shown by charts 1 and 2, the mild and the moderately sick patients do not receive enough benefit to warrant the treatment with antitoxin.

TABLE 1.—*Results Obtained Following Intravenous and Intramuscular Therapy*

Case	Temperature on Admission	Temperature 24 Hours Later	Temperature 48 Hours Later	Days of Illness	Units of Antitoxin	Method Given	Day of Appearance of Serum Rash
1023	103	99	98	4	10,000	Intravenous	6
860	103.6	99	98	3	10,000	Intravenous	0
1178	104.4	99	100.4	3	5,000	Intravenous	0
1324	105	99	99	5	5,000 5,000	Intravenous Intramuscular	0
334	104.2	99.2	99	4	5,000 2,500	Intravenous Intramuscular	0
770	102.4	99.8	99	2	5,000 5,000	Intravenous Intramuscular	0
523	103.8	100	99.4	4	5,000 2,500	Intravenous Intramuscular	0
1324	105	99	99	5	5,000 5,000	Intramuscular Intramuscular	0
1598	105	100.8	100.0	4	10,000	Intramuscular	0
1051	104.8	100	99	2	5,000	Intramuscular	0

TABLE 2.—*Data of Patients in Whom Results Were Not Noted Following Antitoxin*

Case	Temperature: On Admission	Temperature: 24 Hours Later	Temperature: 48 Hours Later	Days of Illness	Units of Antitoxin	Method Given	Day of Serum Rash	
337	103.4	104	104	3	2,500	Intramuscular	0	At the end of 48 hours a second dose was given and the temperature fell to 99 where it remained
1270	103.4	103.6	103.4	3	5,000	Intramuscular	0	In this case a second dose had no effect, the temperature and clinical picture remaining unchanged
952	103.4	102	101.8	2	5,000 5,000	Intramuscular Intramuscular	0	A second dose was again given, and there were no results
2024	103.4	102.0	102.4	3	2,500	Intramuscular	Third day	

Not only does the antitoxin cause the temperature to fall to 100 F. or below within from twenty-four to forty-eight hours, but the entire clinical picture is changed. The rash fades more rapidly, the subjective symptoms disappear, and the patient feels improved. One cannot appreciate this sudden change until one has seen the patient before and after treatment.

2. In those cases in which the patients do not show an improvement after forty-eight hours, a second dose of antitoxin should be given. This fact is clearly shown in case 337 in table 2. The initial dose failed to give the desired results; after the second one, there was immediate improvement, and from that time on the patient had an uneventful recovery. Cases 1270, 952 and others suggest the possibility that several types of streptococci may cause scarlet fever. As suggested by Dr. Park, it is fair to assume that the causative strain in these cases was different from that used to produce the kind of antitoxin employed.

3. Extremely ill and toxic patients should be given the antitoxin either intravenously or intravenously and intramuscularly combined. By giving it this way, immediate results may be obtained. Whether it is to be given by the combined methods or just intravenously must be decided by the case per se, as no set rule can be made.

4. The serum rash is often most distressing and causes more suffering than a moderate attack of the disease, and since there is no way by which one may tell whether there will be a rash, there is another reason why the antitoxin should not be given to patients suffering from mild and moderate attacks. Comparison of the number of patients that develop a rash following intramuscular and intravenous medication shows little variation.

5. Epinephrine and ephedrine were both used to treat patients who had a rash, and it was found that epinephrine gave the best results, although it was often necessary to repeat the dose as often as from every thirty to forty minutes in order to give relief. So great was the discomfort that in some cases morphine had to be given.

6. Scarlet fever antitoxin decreases the number of complications.

7. The exact dosage to be given cannot be stated in a categorical manner, as the severity of the case, the age and the size of the patient must be considered.

8. As in all cases in which antitoxin is given, the sooner it is used, the more immediate will the results be.

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THE SURFACE TENSION OF THE BLOOD PLASMA IN CHILDREN

VARIATIONS IN DISEASE, AND RELATION TO TIME DROP, SEDIMENTATION RATE AND VISCOSITY *

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NORMAL SURFACE TENSION AND VARIATIONS IN DISEASE

A considerable amount of work has been done to determine the surface tension of blood plasma and serum by three or four different methods. The actual results obtained by various authors are for the most part not comparable because of the variety of methods used and therefore the great variation in results. Du Noüy, using his surface tensiometer, has done a great deal of work to determine the time drop of blood serum, using dilutions of 1 : 10,000 with special reference to antibody formation and immunization.

Clausen,[1] using the drop weight method of Hawkins, found a slightly increased surface tension of the blood serum in glomerular nephritis, a decreased surface tension in parenchymatous nephritis and no decrease in cardiac edema. He also noted that eczema causes a slight lowering of surface tension.

Leiter,[2] using the du Noüy surface tensiometer and serum diluted 1 : 100 and undiluted, found that with edema, a low plasma albumin and a high plasma fat there is a low surface tension. This was, however, not always the case.

In the present study, the surface tension was determined on undiluted blood plasma at 21 to 24 C. by the use of two capillary tubes of unequal diameter. This was a slight modification of the method described by De Eds;[3] the results were expressed as dynes per centimeter by use of the formula given by De Eds. The two carefully calibrated capillary tubes held together by a rubber disk were set in a test tube with a flat bottom; this tube contained the plasma to be tested. The plasma was drawn up in the tubes to wet the surface beyond the level at which it would come to rest. The distance between the two menisci was measured

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* From the Division of Pediatrics Stanford University School of Medicine.

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2. Leiter, Louis: The Surface Tension of the Blood Serum in Nephritis, J. Clin. Investigation **3**:267 (Dec. 20) 1926.

3. De Eds, F.: A Simple Method for Measuring Surface Tension Changes of Pure and Biologic Fluids, J. Lab. & Clin. Med. **11**:21 (Feb.) 1926.

by a pair of fine pointed calipers and read on a millimeter scale. This reading in millimeters, by substitution in the formula given by De Eds, was converted into dynes per centimeter. It was found that frequently readings had to be continued over several hours before a constant difference in levels was reached, which was always taken as the final reading.

The results of ninety-five surface tension readings are shown in tables 1 to 7. The mean surface tension in eighty-seven cases (excluding

TABLE 1.—*Surface Tension in Normals*

Diagnosis	Sex	Afebrile Febrile	Age	Dynes per Cm.
1. Normal, chronic tonsillitis	F	A	12	51.48
2. Normal	F	A	13	51.80
3. Normal	M	A	11	51.48
4. Normal	M	A	9	54.02
5. Normal, old poliomyelitis	M	A	10	56.80
6. Normal, eleventh postoperative day	M	A	11	56.07
7. Normal, twelfth postoperative day	M	A	4½	44.07
8. Normal, hernia	M	A	12	49.21
9. Normal, broken arm	F	A	10	52.92
10. Normal, old poliomyelitis	F	A	11	52.92
11. Normal	F	A	9	51.48
12. Normal, moron	F	A	8	52.92

TABLE 2.—*Surface Tension in Kidney Disease*

Diagnosis	Sex	Afebrile Febrile	Age	Dynes per Cm.	Notes
1. Nephrosis with edema	M	A	8	36.73	Lipemia
2. Nephrosis with edema	M	A	10	41.80	Lipemia
3. Nephrosis with edema	M	A	10	47.41	Lipemia
4. Nephritis, acute; no edema	M	A	5	55.05	
5. Nephritis, acute; no edema	M	A	7	51.80	
6. Nephritis, end stage; no edema	M	A	13	38.56	No lipemia

TABLE 3.—*Surface Tension in Rheumatic Fever Group*

Diagnosis	Sex	Afebrile Febrile	Age	Dynes per Cm.
1. Endocarditis	M	F	11	53.62
2. Chorea	M	A	13	50.00
3. Rheumatic fever	M	A	9	55.05
4. Chorea	F	A	12	54.02
5. Endocarditis	F	A	12	51.80
6. Endocarditis and rheumatic fever	F	A	12	50.75
7. Chorea	M	A	14	55.51
8. Endocarditis	F	A	11	56.80

those showing a lipemia) was 52.09 dynes per centimeter, with a normal variation of 5.16 dynes per centimeter. Eighty-eight and five tenths per cent of these cases, however, fell between 48 dynes per centimeter and 55.90 dynes per centimeter. The greatest variation from the mean was seen in cases of kidney disease with plasma lipemia, in which there was a marked lowering of surface tension. This confirms Clausen's observations. In one case there was a low surface tension with no apparent lipemia, and one case of acute glomerular nephritis showed an increased

surface tension. There were two cases of plasma lipemia not associated with any demonstrable lesion in the kidney; in one case, a case of hives, the surface tension was 47.71 dynes per centimeter, which is within the normal variation; in the other case, one of esophageal stenosis, the surface tension was 36.73 dynes per centimeter. The average surface tension of the girls was slightly higher than that of the boys, even when the cases of kidney disease with lipemia were excluded. Five of eight cases of rheumatic fever, chorea or endocarditis showed a surface tension above the mean, but within the limits of normal variation. In twenty-

TABLE 4.—*Surface Tension in Tuberculosis*

	Diagnosis	Sex	Afebrile Febrile	Age	Dynes per Cm.	Notes
1.	Pott's disease 4/24/27	F	A	8	45.91	Same patient
2.	Pott's disease 5/14/27	F	A	8	52.92	
3.	Pulmonary tuberculosis	M	A	4	51.48	
4.	Adenitis, tuberculous	F	A	12	55.05	
5.	Pleurisy with effusion 5/14/27	F	F	35	55.02	Same patient
6.	Pleurisy with effusion 5/18/27	F	F	35	55.51	
7.	Tuberculosis of hip 5/18/27	F	F	13	51.48	Same patient
8.	Tuberculosis of hip 5/31/27	F	F	13	50.32	
9.	Tuberculosis of hip 6/21/27	F	F	13	52.92	
10.	Tuberculosis of hip 7/1/27	F	F	13	51.80	
11.	Miliary tuberculosis	F	F	5	51.48	
12.	Mediastinal gland tuberculosis	M	F	7	49.65	

TABLE 5.—*Surface Tension in Allergy*

	Diagnosis	Sex	Afebrile Febrile	Age	Dynes per Cm.	Notes
1.	Eczema; syphilis, congenital	M	F	½	52.35	
2.	Asthma	M	A	8	51.48	
3.	Eczema	M	A	1	51.80	
4.	Eczema	F	A	2½	53.30	
5.	Asthma, in an attack	F	A	10	51.48	
6.	Hay fever	M	A	4	52.52	
7.	Urticaria	M	A	1½	51.80	
8.	Eczema	F	A	½	52.92	
9.	Asthma, in an attack	F	A	9	53.30	
10.	Eczema	F	A	7	49.99	
11.	Asthma	F	A	10	53.30	
12.	Urticaria	M	A	8½	47.71	Slight lipemia
13.	Asthma	F	A	10	52.20	
14.	Asthma; sinusitis	M	A	5½	51.10	

four febrile cases the average surface tension was 1.5 dynes per centimeter higher than in the seventy-one afebrile cases. A significant or constant deviation from the mean was not found in any other group of conditions. In fourteen cases of allergy (asthma, eczema, hay fever and urticaria) there was a normal average surface tension and the variation was not greater than in the normal group. In the group classed as normal, there was a wide variation in surface tension, from 56.80 dynes per centimeter to 44.07 dynes per centimeter. Excluding the cases showing a lipemia, in the entire group there were more cases showing a reading above the mean than below it. The surface tension is apparently not influenced by the age of the patients.

CORRELATION OF SURFACE TENSION, TIME DROP,—SEDIMENTATION RATE AND VISCOSITY

In making the surface tension determinations as described, it was noted that there was considerable variation in the length of time required for the readings to reach a constant level. This time is analogous to the time drop of du Noüy, but the two methods of determination of surface tension are so dissimilar that the results are not directly comparable.

TABLE 6.—*Surface Tension in Miscellaneous Conditions*

	Diagnosis	Sex	Afebrile Febrile	Age	Dynes per Cm.	Notes
1.	Postdiphtheritic paralysis	F	A	4	48.85	
2.	Psoriasis	M	A	5	53.30	
3.	Abscess of jaw	M	A	6	52.92	
4.	Nephrectomy, tenth postoperative day	F	A	6	49.21	
5.	Thrombopenic purpura	F	A	9	55.05	
6.	Vaginitis	F	A	6	52.35	
7.	Head injury, old	F	A	8	51.48	
8.	Mental instability	M	A	5	52.20	
9.	Mental disorder	M	A	9	46.64	
10.	Abdominal pain, unknown origin	M	A	11	45.92	
11.	Malnutrition	M	A	12	51.80	
12.	Gastro-enteritis, chronic	M	A	12	55.05	
13.	Stenosis, esophagus	M	A	13	36.73	Lipemia
14.	Diabetes	F	A	26	51.48	
15.	Curettage, sixth postoperative day	F	A	26	53.30	
16.	Cervical adenitis, suppurating	F	F	7	49.65	
17.	Syphilis, congenital	M	A	9	52.20	
18.	Syphilis, congenital	F	A	14	51.48	
19.	Pneumonia, lobar; otitis media	F	F	2	51.10	
20.	Pneumonia	F	F	7	51.10	
21.	Pneumonia, convalescent; afebrile, 1 day	M	A	9	51.10	
22.	Fever, unknown origin; afebrile, 1 day	M	A	4	49.21	
23.	Fever, unknown origin; afebrile, 1 day	M	A	4	51.48	
24.	Fever, unknown origin; typhoid (?)	F	F	12	56.07	
25.	Pyelitis	F	A	5	51.10	
26.	Pyelitis	F	F	7	53.30	
27.	Cystitis	F	F	3	55.05	
28.	Hematuria, unknown cause	F	A	9	51.48	
29.	Enuresis, severe persistent	F	A	13	49.65	
30.	Enuresis, cystoscopy 8 hours before	F	A	13	51.80	
31.	Otitis media	F	A	8	54.75	
32.	Sinusitis, acute	F	F	8	53.30	
33.	Sinusitis, chronic	M	A	5	51.48	
34.	Tonsillitis, chronic; sinusitis, chronic	M	F	14	54.02	
35.	Tonsillitis, chronic	M	A	8	53.30	
36.	Bronchitis; tonsillitis, chronic	M	A	6	49.21	
37.	Bronchitis	F	A	6	51.48	
38.	Bronchitis	F	A	7	55.51	
39.	Bronchitis	M	F	5	46.64	
40.	Bronchitis, asthmatic	M	A	8	51.10	
41.	Bronchitis, asthmatic	M	F	8	55.05	
42.	Bronchiectasis	F	F	7	56.80	
43.	Bronchiectasis	M	A	9	47.80	

Constant readings were usually attained in from two to eight hours, but in some cases fourteen hours were required. In many instances, the tubes were set up in the afternoon and readings taken hourly for from six to eight hours, and then not again until morning, after from sixteen to twenty hours, when a constant level had been attained. Not infrequently all the samples would show a temporary increase in surface tension while under observation, as shown by an increased difference between meniscus levels of 0.5 to 1 mm., which was followed after from fifteen to twenty minutes by a return to approximately the former point;

no adequate explanation for this was found. Change in temperature could scarcely be held responsible, as the temperature in the laboratory did not vary more than 3 C. from day to day, and there were no sudden changes. The measurements with the fine calipers checked within 0.3 mm. After reaching the constant level, the readings would remain constant within 0.7 mm. for as long as twenty hours, so that evaporation apparently played little part in the drop. The decrease in surface tension did not occur at a uniform rate. Frequently the readings would remain constant, or show little variation for from one to two hours, and then in a fifteen minute interval there would be a drop of as much as 3 mm.

The decrease in difference of space between the two menisci over a variable length of time will be referred to as the time drop. It is, however, possible, as has been suggested, that the controlling factors may not

TABLE 7.—*Summary of Average Plasma Surface Tensions*

	Condition	Number of Cases	Surface Tension, Dynes per Cm.
1.	Normal	12	52.10
2.	Kidney disease	6	45.21
3.	Rheumatic fever group	8	53.40
4.	Tuberculosis	12	51.90
5.	Allergy	14	51.89
6.	Miscellaneous conditions	43	51.42
7.	Age, 0 to 5 years	21	51.30
8.	Age, 6 to 10 years	33	51.25
9.	Age, 11 years and up	41	51.60
10.	All girls	49	52.39
11.	All boys	46	50.40
12.	All boys less those with nephrosis with lipemia	42	51.30
13.	Febrile	24	52.60
14.	Afebrile	71	51.05
15.	All cases	95	51.43
16.	Mean of all cases except those with lipemia	87	52.09

be identical in the De Eds and in the du Noüy methods, and therefore that the results may not be directly comparable.

For purposes of correlation, the drop in the first two hours was determined and then calculated as the percentage of the total drop. This figure is the time drop referred to hereafter.

A time drop of 100 per cent would indicate that the constant level had been reached in two hours. For correlation between surface tension, time drop and sedimentation rate, fifty-one cases were available; five of these, or 9.8 per cent, had a time drop of 100 per cent; one had a time drop of 0; 18, or 35.3 per cent, had a time drop of 50 per cent or less, and thirty-three, or the remaining 64.7 per cent, had a time drop of over 50 per cent, showing that a rapid fall is more common than a slow one.

The average surface tension of the blood of patients showing a time drop of 50 per cent or less was 51.40 dynes per centimeter; of those showing a time drop of 50 to 100 per cent, 50.55 dynes per centimeter. The five cases with a time drop of 100 per cent had an average surface

tension of 51.82 dynes per centimeter. This indicates that the time drop is little influenced by the original plasma surface tension. A comparatively slow time drop is, however, associated with a slightly higher surface tension (0.9 dynes per centimeter) than a rapid time drop.

TABLE 8.—*Surface Tension, Time-Drop, Sedimentation Rate and Viscosity*

	Diagnosis	Age	Sex	Dynes per Cm.*	Time Drop‡	Sedimentation Rate, Mm.†	Viscosity
1.	Vaginitis	6	F	52.35	100.0	9.0	.....
2.	Syphilis, congenital	½	F	52.35	100.0	3.5	.....
3.	Sinusitis, acute	8	M	53.30	100.0	15.0	.....
4.	Stenosis, esophagus	13	M	36.73	0.0	7.5	.....
5.	Nephrosis with edema	10	M	47.41	74.4	20.5	.....
6.	Syphilis, congenital	9	M	52.20	86.6	2.0	.....
7.	Tonsillitis; sinusitis	14	F	54.02	65.6	1.5	.....
8.	Hematuria	9	F	51.48	66.6	1.5	.....
9.	Eczema	7	F	49.99	83.8	2.5	.....
10.	Asthma	10	F	52.20	88.0	2.0	.....
11.	Adenitis, suppurating	7	F	49.65	78.0	20.0	.....
12.	Tuberculosis, spine	8	F	45.91	76.5	10.5	.....
13.	Tuberculosis, pulmonary	4	M	51.48	100.0	9.0	.....
14.	Pleurisy with effusion	35	F	55.02	39.4	15.7	.....
15.	Tuberculosis, spine	8	F	52.92	86.8	8.0	.....
16.	Bronchitis	7	F	55.51	54.7	11.7	.....
17.	Mental case	9	M	46.64	74.1	1.0	.....
18.	Pleurisy with effusion	35	F	55.10	36.7	18.0	.....
19.	Normal	11	M	56.07	29.6	6.0	.....
20.	Chorea	12	F	54.02	73.5	1.7	.....
21.	Endocarditis	11	M	53.62	35.9	3.0	.....
22.	Pyelitis	5	F	51.10	88.5	2.1	.....
23.	Nephritis, acute	5	M	55.05	16.6	8.0	.....
24.	Normal	4	M	44.07	17.7	1.0	.....
25.	Enuresis	13	F	49.65	100.0	3.2	.....
26.	Bronchitis	5	M	46.64	89.0	15.0	.....
27.	Tonsillitis, chronic	6	M	49.21	88.3	13.0	.....
28.	Postoperative, ten days	6	F	49.21	83.6	11.5	.....
29.	Coryza	12	M	49.21	82.0	12.5	.....
30.	Malnutrition	12	M	51.80	17.3	2.5	.....
31.	Nephritis, acute	7	M	51.80	28.5	23.5	.....
32.	Normal	10	F	52.92	13.5	12.0	.....
33.	Asthma	5	M	51.10	73.4	3.5	.....
34.	Fever of unknown origin	4	M	49.21	32.5	20.0	.....
35.	Postdiphtheritic paralysis	4	F	48.85	92.0	3.0	.....
36.	Tuberculosis, hip	13	F	51.48	39.1	23.0	.....
37.	Poliomyelitis, old	11	F	52.92	87.5	6.5	.....
38.	Pneumonia	9	M	51.10	85.4	19.0	.....
39.	Asthmatic bronchitis	8	M	51.10	65.6	8.0	.....
40.	Endocarditis	12	F	51.80	69.9	9.0	.....
41.	Tuberculosis, hip	13	F	50.32	79.3	23.0	.....
42.	Bronchitis	6	F	51.48	45.4	12.0	1.729
43.	Psoriasis	5	M	53.30	28.5	9.0	.....
44.	Mental case	5	M	52.20	22.6	11.5	.....
45.	Tuberculosis, hip	13	F	52.92	17.5	21.0	.....
46.	Nephritis, end stage	13	M	38.56	69.0	14.0	1.583
47.	Normal	9	F	51.48	63.6	3.5	1.649
48.	Abscess of jaw	6	M	52.92	88.5	13.0	1.924
49.	Tuberculosis, miliary	5	F	51.48	14.9	15.0	1.398
50.	Pneumonia, lobar	2	F	51.10	36.8	21.5	1.885
51.	Tuberculosis, hip	13	F	51.80	35.0	19.5	1.830

* Surface tension expressed in dynes per centimeter.
‡ Time drop—drop in the first two hours expressed as the percentage of the total drop.
† Sedimentation rate expressed as the distance in millimeters the red cells have fallen in two hours.

In the fifty-one cases, the sedimentation rate was determined on part of the same sample of citrated blood from which plasma was obtained for the surface tension determination. Nine-tenths cubic centimeters of blood was added to 0.1 cc. of 1 per cent sodium oxalate solution in a small graduated tube, and the height of the column of plasma above the level of the cells was read every five minutes for seventy-five minutes.

In order to compare the sedimentation rate and the time drop, the time drop expressed as percentage was plotted on the axis of ordinates, and the sedimentation rate, expressed as the distance in millimeters the red cells had fallen in thirty minutes, was plotted on the axis of the abscissas. No correlation was found between the two factors.

The average plasma surface tension in all cases showing a slow sedimentation rate (here taken as those in which the red cells had settled 15 mm. or less after thirty minutes) was 50.07 dynes per centimeter. Those in which the cells had settled beyond 15 mm. within thirty minutes, a comparatively rapid sedimentation rate, had an average surface tension of 51.2 dynes per centimeter. Thus a rapid sedimentation rate is associated with a slightly higher plasma surface tension than is a slow or more normal sedimentation rate.

In seven cases, the viscosity of the blood plasma was also determined. This was done by comparing the time required for the plasma to run out of a given length of glass tubing with the time required for water to run through the same tube. It was impossible to demonstrate any influence of the viscosity on the time drop, the sedimentation rate or the surface tension in this very small series. It is interesting to note, however, that the lowest and highest viscosities obtained showed nearly the same surface tensions.

The results here discussed were compiled from the material given in table 8.

SUMMARY AND CONCLUSIONS

1. The mean blood plasma surface tension in eighty-seven determinations (those showing plasma lipemia excluded) was 52.09 dynes per centimeter, with a normal variation of 5.16 dynes per centimeter; 88.5 per cent of these cases fell between 48.00 dynes per centimeter and 55.90 dynes per centimeter.

2. The most marked and consistent lowering of surface tension occurred in cases of kidney disease with lipemia.

3. Allergy did not appear to cause any constant variation.

4. In eight cases of rheumatic fever, chorea and endocarditis there was an average surface tension slightly above the mean.

5. Fever apparently caused a slight rise in surface tension.

6. The surface tension in girls appeared to be slightly higher than in boys.

7. Age probably does not have any influence on surface tension in children.

8. There was no correlation between the sedimentation rate of the red blood cells and the time drop of the plasma.

9. The time drop was influenced little, if at all, by the surface tension. With a rapid time drop, however, the surface tension was slightly lower than with a slower time drop.

10. With a rapid sedimentation rate, the surface tension was slightly higher than it was with a slow sedimentation rate.

11. In seven cases, there was no apparent influence of the viscosity on the sedimentation rate, time drop or surface tension.

AN ANALYSIS OF ANOREXIA*

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Information regarding the appetite of children is notoriously absent from the medical literature. The analysis, classification and treatment of other such prominent symptoms as glycosuria, convulsions and dyspnea have always held an important position in medical textbooks and journals. There are many reasons why the symptom "anorexia" has been neglected. A child's appetite is normally good, and when it is depleted it becomes a secondarily subjective symptom through the objective observation of the mother. There are few dependable objective signs that will infallibly confirm or deny the subjective complaint of loss of appetite in children. As far as this report is concerned, anorexia is considered unimportant unless it is severe and chronic enough to produce objective signs of malnutrition. There is such a variety of methods of treatment for a diminished appetite in the absence of any definite and specific stimulant that it is doubtful whether or not they have any value. The fact remains and is obvious to those in close contact with children that the problem of appetite is an important one.

Anorexia in children is not a specific disease entity but at best is only a symptom of organic or functional disorder. This study was undertaken with the object of determining the most common pathologic conditions underlying a definite chronic anorexia, the relative importance of anorexia as a presenting symptom and the most efficient and reliable method of treatment. At the start, anorexia was defined to mean a subjective complaint of constant loss of appetite persisting long enough to cause objective signs of malnutrition. It was then found necessary to subdivide these cases into two groups: (*a*) organic anorexia in which a definite possible cause for the condition could be determined, and (*b*) functional anorexia, in which an organic cause that could possibly explain the condition was not found. It is with the latter group that this paper is largely concerned.

In order to determine the approximate incidence of anorexia in children, the consecutive records of 1,471 children from the outpatient department between the ages of 1 and 14 years were carefully inspected in regard to the subjective and objective symptoms. This group of cases represents children coming to the outpatient department between July

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and the following February. Only children with loss of appetite as a presenting symptom and who on examination showed signs of malnutrition confirming such a symptom were included in this study. The fact that the physical condition of the patient confirmed the complaint of anorexia was found to be highly important because of the frequent complaint by apprehensive mothers that abundantly well-nourished children "hadn't eaten anything for weeks." Children who had apparently lost their appetite because of excessive indulgence in sweets and food between meals were seldom found to show signs of malnutrition and were consequently excluded as far as possible from this study.

In adult patients, probably the most stubborn and resistant anorexia is that which almost invariably accompanies severe anemia. Certainly there is a strong relationship between the condition of the blood, particularly the hemoglobin and the red blood cells, and the proper function of the appetite. It is also common experience that when the severe anemia diminishes, because of any form of treatment, the first and most promising sign of improvement is the return of the appetite. This is particularly true in pernicious anemia in which the return of the appetite always marks the beginning of remissions. The return of the appetite, sometimes exaggerated, is common following transfusion in severe anemia of either the primary or the secondary type. When liver diet is used in the treatment of patients who have pernicious anemia, it is striking how soon the appetite improves even in cases in which the anorexia is so pronounced that the feeding of liver has to be initiated by gavage feedings. As this observation was significantly frequent, it was suggested, first, that liver in itself might be a specific stimulant to the appetite when fed to malnourished children and, second, that the loss of appetite in poorly nourished children might often be due to a mild anemia that had hitherto been overlooked or ignored. In cases of functional anorexia that has persisted for some time, a mild anemia is frequently present, and it is impossible to say whether the children are anemic because of their loss of appetite or whether they have lost their appetite because they are anemic. Nevertheless, it is relatively unimportant which of these factors is primary, providing the two conditions can be corrected simultaneously. Reasoning *a priori*, there are many facts which make a liver diet a good one for undernourished children, because of the vitamin content, the nature of the protein contained and the distinct effect of liver, beefsteak and kidneys in producing hemoglobin.

RESULTS OF DIETS

Table 1 is a classification of the presenting symptoms of 1,471 children from the outpatient department grouped in order of their frequency. Of the 1,471 children, 349 or 24 per cent, came to the hospital with loss of appetite as the primary presenting symptom. Its

important position is easily seen when it is compared with the common cough or cold. It is also worthy of note that nearly one half of the children brought to the hospital were in need of dietary instructions rather than any form of medication. That is, 40 per cent came to the hospital with the complaint of loss of appetite, pallor, underweight or overweight, and regulation of feeding.

TABLE 1.—*Incidence of Certain Symptoms in Children Seen in the Outpatient Department from a Series of One Thousand Four Hundred and Seventy-One Consecutive Cases*

	Complaint	No. of Cases		Complaint	No. of Cases
1.	Anorexia	349	26.	Obesity	10
2.	Cough or cold	118	27.	Weakness	8
3.	School examination	110	28.	Discharge from eyes	8
4.	Pallor	96	29.	Bowleggedness	7
5.	Underweight	79	30.	Backwardness	6
6.	Abdominal pain	68	31.	Epistaxis	6
7.	Skin rash	65	32.	Jaundice	5
8.	Swelling of parts	64	33.	Insomnia	5
9.	Nervousness	56	34.	Hoarseness	5
10.	Sore throat	55	35.	Cyanosis	4
11.	Vomiting	41	36.	Tenesmus	4
12.	Regulation of feeding	40	37.	Frequency	4
13.	Enuresis	29	38.	Constipation	4
14.	Dyspnea	24	39.	Pruritus	4
15.	Dental caries	24	40.	Talking or walking in sleep	3
16.	Vaccination	22	41.	Bloody urine	3
17.	Paralysis	19	42.	Pus in urine	3
18.	Pain in joints	17	43.	Swollen ankles	3
19.	Fever	17	44.	Dysuria	3
20.	Diarrhea	16	45.	Swelling of abdomen	2
21.	Convulsions	15	46.	Urolapse of rectum	2
22.	Pain in chest	15	47.	Sweating of head	2
23.	Deafness or earache	14	48.	Nausea	2
24.	Headache	12	49.	Large head	2
25.	Adherent prepuce	11	50.	Fracture	1

TABLE 2.—*Classification of the Conditions Most Commonly Found Associated with Marked Anorexia in Three Hundred and Forty-Nine Cases*

	Condition	No. of Cases		Condition	No. of Cases
1.	No lesion to account for condition	121	10.	Rickets	7
2.	Septic tonsils and adenoids	77	11.	Bronchitis	6
3.	Acute infectious diseases	29	12.	Catarrhal jaundice	6
4.	Tuberculosis	24	13.	Intestinal parasites	3
5.	Pyelitis	23	14.	Scurvy	2
6.	Dental caries	15	15.	Abscess of the lungs	1
7.	Heart disease	13	16.	Splenic anemia	1
8.	Constipation	11	17.	Bronchial asthma	1
9.	Chorea	9			

Table 2 gives a classification of the pathologic conditions that were allowed as possible causes in this group of 349 malnourished children with subjective anorexia. In 121 cases, or 35 per cent, no lesion was found that could account for the patients' condition, and these were accordingly grouped as cases of functional anorexia. Patients who had not had tuberculin tests and whose chests had not been examined by the roentgen ray to rule out of consideration a possible tuberculosis were not included in this group. The most common organic cause of anorexia

in this series was septic tonsils and adenoids. Among the other common causes were acute infections, tuberculosis and pyelitis.

In an attempt to determine the most efficient method of treatment in the cases of functional anorexia use was made of: (1) daily administration of saccharated iron, (2) daily administration of cod liver oil and (3) the introduction of fresh calf's liver, broiled beefsteak and lamb's kidneys into the diet at least three times a week. The first treatment supplied iron, the second vitamins and the third provided both and a food with a high protein value.

It is interesting that in this series of cases there were only two children who had ever been fed liver in any form. The general impression is that children are rarely encouraged to eat liver, beefsteak or kidneys and that parents are frequently surprised to hear the physician prescribe such foods for children. However, there is no reason to think that such foods are not suitable for the diet of normal children. Children with functional anorexia, as the term is here interpreted, were required to show some definite objective signs of the condition. Among the forty cases presented, all patients showed either a failure to gain weight or a loss of weight during the three months prior to the institution of treatment. Most of them showed a mild anemia in addition, which in many instances consisted largely of a lowered hemoglobin content, and, in fewer instances, of an actual diminution of the red blood corpuscles. As the objective criterion of the appetite the body weight was chosen primarily because of its reliability. Fundamentally, a child with loss of appetite will not gain weight unless some organic disturbance, such as edema, is present. In normal children, an increase in body weight after a period of failure to gain certainly can be attributed to increased ingestion of food. As far as can be ascertained, there is no other acceptable objective criterion of the appetite of children. In this study the records of the body weight prior to treatment and subsequent to treatment afford the best basis of comparison. In most cases, the records of the body weight were available for at least three months prior to treatment and for at least three months subsequent to the initiation of treatment. All weights were taken under standard conditions, without clothing and on the same scale. The weights were all recorded in pounds, and the accuracy of one-half pound was used as a statistical unit in making comparisons.

The choice of treatment in certain cases was in part accidental and in part due to individual variation in taste. It was found impossible to feed cod liver oil to certain children, and a few of these are included in the group given 5 grains (0.3 Gm.) of saccharated iron three times a day. Fourteen children refused to eat the liver, and a few of these are included in the group given ½ ounce (15 cc.) of cod liver oil two or three times a day. In general, there was not much difficulty in feed-

ing these children the liver diet, providing some time was spent in teaching the mother how to prepare the liver. It was found convenient in some cases to withhold other food until the liver or kidney had been eaten. The majority of the children enjoyed the liver, perhaps in part because it was so far removed from the ordinary food fed to children and perhaps also because it was an unusual prescription to be given by a physician. The children even enjoyed taking the prescription to the butcher rather than to the druggist.

In some of the more striking cases, distinct anemia was associated with malnutrition. In several of these cases samples of the blood were taken frequently for the determination of hemoglobin and red cells. It was found stimulating to the children to watch the increase in body weight and hemoglobin with each visit to the hospital, while they steadily improved in appetite and bodily vigor on the liver diet. The improvement was distinctly less noticeable in the children given cod liver oil or saccharated iron only.

TABLE 3.—*Results of Treatment with Saccharated Iron in Functional Anorexia*

Case	Age	Sex*	July	August	September	October	November	December	January	February	Total Gain
1	5	♂	36 36	35.5 35	36 34.25	35 |	37	37.5	38 38	37.5	2.5
2	7	♀	44	44	44	44 |	46.5	46.5	47	48	4.0
3	4	♂	29	31	30.25	30 |	32 32 33		33.5		3.5
4	9	♂	53	53	52.5	53 |	56	56		56	3.0
5	8	♀	51	51	50.25	50 |	52	54		57	7.0
6	5	♂	37	34.5		34 |	35.25 36	35.5		36.5	2.5
7	6	♀	41	43.75 42	42 42.5	42 |	44.25	43		45	3.0

* In this table and in tables 4 and 5, ♂ indicates male; ♀, female.

The results of treatment are shown in tables dated from the first of July to the last of February, including the three months prior to treatment and the four months subsequent to treatment. The difference between the body weight at the beginning of treatment and at the end of the observation is taken as the total gain in weight. The vertical broken line through the middle of the tables indicates the time of initiation of treatment. The time of the various weighing is shown as nearly as possible by the horizontal position of the figure. More frequent weighings were made in some instances than in others, and all the figures are not given because of their frequency or insignificance.

Table 3 shows the results of treating seven children with functional anorexia with 5 grains of saccharated iron three times daily over a period of several months. A general improvement was noted in some instances, while in others the results were not striking. The range of total gains has so wide a dispersion and the number of cases is so small that the probable error is great and does not justify a conclusion in regard to the efficacy of saccharated iron.

Table 4 shows the results of treating nine patients who had functional anorexia with one-half an ounce of cod liver oil two or three times daily over a period of several months. A general improvement was noted in every case. The range of total gain here likewise has such a wide variation that with so small a group of cases a conclusion cannot be drawn in regard to the value of the treatment.

Table 5 shows the effect of treating twenty-four patients who had functional anorexia with a diet containing liver, beefsteak and kidneys at least three times a week. Here the results are striking in the majority of cases. Children who were markedly undernourished and pale became robust and healthy, and there was an immediate increase in the appetite in every case. In many cases the clinical improvement exceeded any objective criterion available. The range of total gains is still wide, but

TABLE 4.—*Results of Treatment with Cod Liver Oil in Functional Anorexia*

Case	Age	Sex	July	August	September	October	November	December	January	February	Total Gain
8	6	♂	42	42		40	44	43.5		43.5	3.5
9	4	♀	28	28.5 28		27	29 31	31.5 33		36.5	9.5
10	3	♂	30 29	29.5	30.5	29	31 31.5	33.75 34		35	6.0
11	7	♀	46	46.5 46		46	48.5			50.25	4.25
12	10	♂	58	58 57		57	57.5	60.25	59.75	60	3.0
13	5	♂	38	38		37.5	40.25	42		42	4.5
14	8	♂	50 49		47.25	47	49 49 50	50.75		52	5.0
15	12	♀	64.5	63	63.75	63	62.5	63	64		1.0
16	11	♂	61	62		61	63	60		67	6.0

the dispersion of results is not as great as in either of the two foregoing groups and the number of cases is larger. The probable error in this group is less than half that of the second group and nearly half that of the first group.

From a statistical standpoint, the first two groups cannot be compared with one another because the difference of the means is only 1.66 times the square root of the sum of the squares of the probable errors of the two groups. Either the first or the second group of cases can be compared with the third group, however, because the probable error is then within the realm of statistical significance. When the second and third groups are compared, the difference of the means is 4.56 times the square root of the sum of the squares of the probable errors. Therefore, conclusions drawn between the two groups are justifiable. The mean total gain in weight in the second group, the children treated with cod liver oil (table 4), is 4.75 ± 0.634 pounds; the mean total gain in weight in the third group, the children treated with the liver diet (table 5), is 7.90 ± 0.276 pounds. It is obvious from this comparison that the children fed on liver showed the best results.

When the first and third groups are compared, the difference of the means is 8.13 times the square root of the sum of the squares of the probable errors. These groups, therefore, offer the best opportunity for comparison beyond the possibility of chance sampling. Here the first group, the children treated with saccharated iron (table 3), show a mean total gain in weight of 3.50 ± 0.402 pounds; the third group, those treated with the liver diet (table 5), show a mean total gain in weight of 7.90 ± 0.276 pounds. Here, likewise, it is conclusive that the best results were obtained with the use of the liver diet.

TABLE 5.—*Results of Treatment with Liver, Beefsteak and Kidney Diet in Functional Anorexia*

Case	Age	Sex	July	August	September	October	November	December	January	February	Total Gain
17	5	♂	39	39.25	38.5	37	37.5 38.5	39.5	41.75	43	6.0
18	8	♀	49 49	47.5		47	48.5 49 49	50	52	54 56.5	9.5
19	12	♀	69.5	67		64	67.5 67.75	68	69.75	71	7.0
20	10	♂	58	58		57.5	59.75 61.5 62	63.75	67.5	69.5	12.0
21	8	♂	48 48 48.5	48.5 48.5	48	48.5	49 53.5	55		55	6.5
22	7	♂	45 44	46.5 44.25		43	48.5	49	49		6.0
23	9	♀	54	54.25	54.25	54		58.25	60	61	7.0
24	4	♂	37 37.5	38 37.5	37 36.5	37	39.5	41.25 42.50	44	46.5	9.5
25	6	♂	43 43	43 42	42	42		47.5	48	48.75	6.75
26	5	♀	36 37	37.5	36	35	39	40.5	41	43	5.5
27	11	♂	57 57	56	56	55.75	56.5	57 59	59.5	62.5	7.0
28	13	♂	72	70.25	71	70.5	72	73.25	75.25 76.75	78	7.5
29	3	♀	26.5	25.75	26	26	27.75	28	29.5	31	5.0
30	6	♂	41.5 41	41		40.25		45	45.5	48	7 75.
31	8	♂	56 56	56 56.75	56.5	56.75	56.5	57 58	58 59	59.5	3.75
32	9	♂	60 60	61 60.75	60	60.5	63	64	64.5	66.5	6.0
33	7	♂	42 42	42		42	45.5 46	47.25	49 49	49	7.0
34	6	♀	39 39.5	38 38		38	41 41.5	44	46	47.5	9.5
35	4	♀	34	34	33.5	34.5		37.5	37.75	38.5	4.0
36	9	♂	52	53.75		52	55	55.5	58 59	62	10.0
37	11	♂	56.5	54 54.25		54		58.5	59	60.5	6.5
38	12	♀	57 57	56.5		57	61.75	62.5	63	64	7.0
39	8	♂	52.5 53	53 53	53 52.5		57	58.5	59.25	62	9.5
40	11	♀	57 55.5	53.5	53.5	54	57.5	57	57.5	58	4.5

COMMENT

Most of the literature concerned with the study of the appetite of children heretofore has dealt with functional anorexia of the specific type called "anorexia nervosa." This term has been purposely ignored in this paper, because I do not believe that more than a few of these cases could be properly classified under that heading and that the term functional anorexia is more inclusive and safer, and does not suggest the existence of any underlying nervous condition unwarranted by the absence of definite neurologic stigmas. Among the more recent papers

of this type are those by St. Engel [1] and Myers,[2] who emphasized properly the importance of anorexia in children's clinics, but who write in a general manner about anorexia nervosa and suggest reassurance and changed environment as the best therapeutic procedures.

In a paper on the treatment of anorexia in children in the recent literature, Hobhouse [3] stated that the child who has lost inclination for food will always be one of the most common problems of the practitioner. He likewise classified the cases of anorexia into a large group in which a cause cannot be found; a small group in which a definite cause, such as scurvy and tuberculosis is found; a large group in which anorexia seems to be due to dietetic errors, such as diets poor in protein, poor in fat or excessive in carbohydrate; he mentioned a small group in which the anorexia seemed to be due to a definite neurosis. In regard to treatment, Hobhouse suggested mainly cod liver oil, the administration of iron and environmental change.

In 1919, Benedict, Miles, Roth and Smith [4] in their studies of human vitality and efficiency under prolonged dietary restriction showed conclusively that incomplete diets, particularly those low in protein and high in carbohydrate, can lead to anemia. This was further substantiated by the work of McCarrison.[5] In 1918, Pearce, Krumbhaar and Frazier [6] published a review of the early experimental work concerning the effect of certain types of food on blood regeneration. In 1922, Jencks [7] showed that an abundance of vitamins in the diet favored blood regeneration. McCollum [8] mentioned that the feeding of liver and kidneys can enhance remarkably the growth of animals. In 1926, Minot and Murphy [9] published their results in feeding a special diet rich in liver, kidneys and beefsteak to patients with pernicious anemia, and

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1. St. Engel: Loss of Appetite in Childhood, Monatschr. f. Kinderh. **30**:270, 1925.

2. Myers, Bernard: The Treatment of Anorexia Nervosa in Children, Practitioner **113**:351, 1924.

3. Hobhouse, Neill: The Treatment of Anorexia in Children, Lancet **1**:10 (March 7) 1925.

4. Benedict, F. G.; Miles, W. R.; Roth, P., and Smith, H. M.: Human Vitality and Efficiency Under Prolonged Restricted Diet, Publication 280, Carnegie Institute of Washington, 1919, p. 364.

5. McCarrison, Robert: Faulty Food in Relation to Gastro-Intestinal Disorder, J. A. M. A. **78**:1 (Jan. 7) 1922.

6. Pearce, R. M.; Krumbhaar, E. B., and Frazier, C. H.: The Spleen and Anemia, Philadelphia, J. B. Lippincott Company, 1918.

7. Jencks, Z.: Studies in Regeneration of Blood, Am. J. Physiol. **59**:240 (Feb.) 1922.

8. McCollum, E. V.: The Newer Knowledge of Nutrition, New York, The Macmillan Company, 1923.

9. Minot, G. R., and Murphy, W. P.: The Treatment of Pernicious Anemia by Special Diet, J. A. M. A. **87**:470 (Aug. 14) 1926.

they noted the following marked effect on the patient's appetite: "In fact frequently they soon became ravenously hungry and often anxious to eat more than the customary allowance of liver and meat." They noted that before the blood changes could be found the appetite improved and the diarrhea disappeared.[10]

Whipple[11] and his co-workers, experimenting with an artificially-produced secondary anemia from hemorrhage in dogs, studied the comparative hemoglobin-producing effect of various types of food and found that liver, kidney and muscle meat had the most pronounced beneficial effect on blood regeneration, which was particularly striking when compared with the dairy products. Following the clinical results of Minot and Murphy,[12] Nicholls[13] reported, in a practical fashion, the various methods of preparing a diet rich in liver and kidney and discussed the difficulties encountered and how these could best be surmounted.

The progress of these investigations has led to the suggestion that the liver diet acts as a stimulant to the regeneration of blood, and this is noted clinically by the return of the appetite. In the absence of any organic disease, anorexia of long standing is frequently found associated with a mild secondary anemia, probably of the nutritional type and sometimes severe. The improvement of the appetite and the general condition following the introduction of the liver diet are striking in these cases. These clinical results obviously suggest that the liver diet contains some specific substance[14] which increases an appetite that is depleted and which renews one that is lost. If this is true, judging from the results that are here reported, it is suggested that this specific substance that produces appetite is present in liver and absent in saccharated iron and cod liver oil. In other words, the factor that produces appetite is probably neither iron nor vitamin D, unless it is the favorable combination of the two which occurs in a more available form in liver.

10. Murphy, W. P., and Minot, G. R.: A Special Diet for Patients with Pernicious Anemia **195**:410 (Aug. 26) 1926.

11. Whipple, G. H.: Blood Regeneration in Severe Anemia: Influence of Striated and Smooth Muscle Feeding, Am. J. Physiol. **79**:260 (Jan.) 1927; Blood Regeneration in Severe Anemia; Influence of Kidney, Chicken and Fish Livers and Whole Fish, ibid. **79**:271 (Jan.) 1927; Blood Regeneration in Severe Anemia; Influence of Dairy Products on Hemoglobin Production, ibid. **79**:280 (Jan.) 1927.

12. Minot and Murphy (footnote 9). Murphy, W. P.: The Use of Liver Diet in the Treatment of Pernicious Anemia, Am. J. Pub. Health **17**:91 (Jan.) 1927.

13. Nicholls, E. A.: Suggestions for the Administration of the Minot and Murphy Special Diet for Pernicious Anemia, Boston M. & S. J. **196**:302 (Feb. 24) 1927.

14. No doubt this substance will be found in the new liver extract being investigated by the Boston Pernicious Anemia Commission and manufactured by the Eli Lilly Co.

SUMMARY

1. Anorexia is defined as a subjective complaint of constant loss of appetite persisting long enough to cause objective signs of malnutrition.

2. Cases are divided into two groups: (*a*) organic anorexia in which a definite possible cause can be determined and (*b*) functional anorexia, in which an organic cause cannot be found.

3. In 1,471 children seen consecutively in the outpatient department 349, or 24 per cent, came to the hospital with loss of appetite as a presenting symptom. One hundred and twenty-one, or 35 per cent, of these children had no discoverable lesion to account for their condition. These cases were classified as functional anorexia.

4. The five commonest reasons for bringing children to the outpatient clinic were: (1) loss of appetite, (2) cough or cold, (3) physical examination for admission to public school, (4) pallor and (5) underweight.

5. The commonest pathologic conditions found associated with marked loss of appetite were: (1) septic tonsils and adenoids, (2) acute infectious diseases, (3) tuberculosis, (4) pyelitis and (5) dental caries.

6. Patients with functional anorexia were treated by three methods: (1) daily administration of saccharated iron, (2) daily administration of cod liver oil and (3) the introduction of liver, beefsteak and kidney into the diet at least three times a week. These methods of treatment are compared.

7. The body weight was found to be the most reliable objective criterion in the treatment of functional anorexia.

8. Although a comparison could not be drawn between the children treated with saccharated iron and those treated with cod liver oil, there was a good opportunity, statistically, to compare both of these groups with the children treated by the liver diet.

CONCLUSIONS

1. More than one third of the children with chronic anorexia do not have an organic pathologic condition underlying the symptom.

2. The most common pathologic conditions found associated with chronic anorexia are septic tonsils and adenoids.

3. Anorexia far exceeds all other presenting symptoms in its incidence in children seen in the outpatient department.

4. The most efficient and reliable method of treatment for chronic functional anorexia is the introduction of fresh calf's liver, broiled beefsteak and lamb's kidneys into the diet of the child.

The feeding of liver may be a specific stimulant to the appetite.

A NEW TEST FOR HEARING IN THE NEW-BORN

THE CONDITIONED REFLEX *

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In very young infants, all tests for hearing have been dependent on visible response of the baby to sounds. This response in the baby has, presumably, been dependent on the inherent fear of sudden or unusual sounds noted in practically all infants. This reaction usually consists in a sudden start of the entire body, a winking of the eye, a crying out, or a combinaion of these activities.

Recently I was asked to pass on the ability of a baby to hear because she failed to respond to this stimulus of sudden noise. A few days previously I had examined this 3 months old baby at The Cradle in Evanston, from which institution she was to be adopted by a patient of mine. The baby appeared to be an exceptionally healthy, bright youngster free from all physical defects, and her mother had a negative Wassermann reaction in the blood. Following my recommendation she was taken into the family, but adoption papers were not immediately signed. After a few days' residence, the foster mother, who had previously adopted a normal boy now 4 years of age, reported that the new baby did not respond to noises as had her other child. She was sure that this baby was deaf, because she did not respond in any way when a door was slammed, when the hands were loudly clapped close by her head, or even when a dishpan at her ear was banged with a spoon. On examining the child in my office the next day, neither I nor a competent otolaryngologist, Dr. H. C. Ballenger, was able to elicit the slightest reaction to sound, though we tried all the different tests that we were able to make.

The baby was returned to The Cradle for further observation. There she was seen by another ear specialist, Dr. Thomas Galloway, who also failed to get any response to sound. After a few days' observation, one or two of the nurses thought they had seen the baby start at sounds, and on one occasion when I clapped my hands when the infant was fast asleep, she moved her arms. Pending further developments, I advised that the adoption be delayed indefinitely. It was the opinion of all concerned, however, that the baby was probably not deaf. This was based on the apparent brightness of the child, the fact that she made the usual articulate sounds for her age, and the

* Received for publication, Sept. 20, 1927.

normal appearance of the visible external and internal ear. Before committing ourselves, however, we all wanted to wait for demonstrable proof.

At this juncture it occurred to me that by establishing a conditioned reflex with sound as one stimulus and pain as the other, we might be able to prove the presence of hearing. Accordingly, at half hour intervals during the next night a small dinner bell was rung beside the baby's crib in such a way that she could not see it, while at the same time the sole of the right foot was firmly scratched with a pin. When these stimuli were applied, the child cried out lustily and drew up the right leg. By mid-morning, after perhaps twelve or fifteen applications, the infant cried and drew up the leg when the bell was rung and *the foot was not touched.* No one could see the experiment without being perfectly sure that the child heard. If the bell was slightly tinkled, her face at once looked worried. The worried look was followed by a cry when the bell was loudly shaken.

On the strength of this evidence I recommended adoption. The infant was adopted, and after a month or so in the home, the mother says that she is sure the child hears and that she is "afraid she is making a nervous wreck of her trying to prove it."

In proposing this as a test for hearing, it seems to me that a similar proceeding might well be instituted in investigating the presence or acuteness of other senses, such as seeing. By some such method it may be possible to investigate the whole subject of sensation in the new-born more accurately. Accordingly, Dr. Beatrice Hawkins proposes to make such investigation during the next few months with the new-born infants at The Cradle.

545 Lincoln Avenue.

EFFECT ON THE URINE OF ADDITION OF ACIDS AND ALKALIS TO THE DIET OF INFANTS*

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Because of the marked buffer action of cow's milk and its consequent inhibitory action on hydrochloric acid, various methods have been used to promote the efficacy of the gastric juice in normal infants, as well as in those with digestive disturbances. Such aids are milk acidified with tenth-normal hydrochloric acid, lactic acid, lemon juice and bacterial cultures. Good results have been obtained with these additions, namely, a steady gain in weight and an improvement in digestive disturbances. Little attention, however, has been paid to the changes resulting in the urine when acid has been added to the milk.

Von Bernuth and Duken [1] observed a series of spasmophilic and non-spasmophilic infants who were given 260 cc. and 400 cc. tenth-normal hydrochloric acid to a liter of milk. In fourteen of twenty-three infants, or 61 per cent, casts developed in the urine; most of these were finely granular, although there was an occasional hyaline cast; sometimes there was a trace of albumin. These urinary changes disappeared rapidly when the acid was omitted from the diet.

Feuchtwanger and Lederer [2] showed that the most important condition for the appearance of casts in the urine is an acid reaction of the urine. When a definite acidity is reached, casts appear. Freudenberg [3] observed that after additions of ammonium chloride a strongly acid urine with casts resulted; these pathologic factors disappeared when this substance was omitted.

The present investigation embraces an extensive study of the urinary changes in infants and in older children who received one of the following acids or alkalis in the milk ingested: tenth-normal and thirtieth-normal hydrochloric acid, lactic acid, lemon juice, sodium bicarbonate or sodium hydroxide in varying quantities and concentrations. A small number of children was selected in order that the effect of these various substances could be observed on the same child for a long period.

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* Received for publication, Aug. 13, 1927.

* From the Home for Hebrew Infants (service of Dr. Alfred F. Hess).

1. Von Bernuth, F., and Duken, J.: Klinische Beobachtungen über Stuhlbeschaffenheit und Zylindrurie bei Salzsäuremilch, Arch. f. Kinderh. **80**:21, 1926.

2. Feuchtwanger, A., and Lederer, M.: Zur Frage der Entstehung von Harnzylindern bei Saüglingen, Jahrb. f. Kinderh. **112**:7, 1926.

3. Freudenberg, E.: Einflusz der Ionen auf die Diurese beim Saügling, Ztschr. f. Kinderh. **39**:608, 1925.

Since 1924, when Hess [4] reported the good results obtained with "lemon-juice-milk," all the infants in one ward at this institution (about twenty-five) have received lemon juice in a dilution of 3 per cent mixed with their milk formula. Eight normal infants were selected for study from this ward in order to ascertain whether the acidity of lemon juice leads to any abnormalities in the urine. Examinations of many specimens of urine showed that the reaction was acid and that abnormal chemical or microscopic changes had not occurred. The hydrogen ion concentration of the urine of three infants for a period of six days showed a range from 5.5 to 7.[5] It is evident, therefore, that the citric acid contained in the 3 per cent dilution of lemon juice did not irritate the kidneys in any way.

The investigation was then continued with three normal infants, aged 7½, 8 and 11 months, whose urine had been normal on several occasions; one of these had been receiving "lemon-juice-milk." Two hundred and forty cubic centimeters of thirtieth-normal hydrochloric acid was added to the milk formula. For the first few days, changes were not noted in the urine. After from two to six days, microscopic examinations showed red blood cells and many finely granular and occasionally hyaline casts in the urine of all three infants; coarsely granular casts were found in two instances and a faint trace of albumin in one. When the acid was omitted from the diet, the urine became normal from three to six days later. As was to be expected, the greater the number of casts, the more frequently granular casts appeared in the urine. In fact, as many as ten casts were not observed in any instance when only hyaline casts were present. In other words, when + + + or + + + + are designated in table 1, granular as well as hyaline casts are indicated. It should be added, however, that not infrequently granular casts appeared when the total number was less than ten, that is to say, when the change has been designated as + +. It was surprising that with the development of casts in the urine, showers of them in some instances, albumin was so rarely observed; even when red blood cells were present, they were rarely noted. Apparently casts, usually the hyaline variety, are the first indication of irritation of the kidney.

To the milk given an infant, aged 3 months, who had tetany, 240 cc. of tenth-normal hydrochloric acid was added. Twenty-four hours later, the urine showed a faint trace of albumin and a few finely granular

4. Hess, A. F., and Matzner, M. J.: The Value of Milk Acidified with Lemon Juice, J. A. M. A. **82**:1604 (May 17) 1924.

5. The p_H of the urine was determined by means of the colorimetric method. For the upper range of hydrogen ions (from 6.6 to 8.5) phenolsulphonphthalein was used. For the concentrations below 6.6, special standards were prepared, for p_H 4 (brom phenol blue), for p_H 5 (brom cresol green) and for p_H 6, (brom phenol red).

TABLE 1.—*Appearance of Casts Following the Addition of Varying Strengths of Hydrochloric Acid to Milk* *

Name.....	S. F.	B. K.	H. W.	M. M.	E. R.	N. F.	A. W.	C. W.	M. F.
Age.....	11 mo.	8 mo.	7½ mo.	3½ mo.	4½ yr.	4 yr.	5 yr.	4 yr.	4 yr.
3/ 3/27.....	240 cc. N/30 HCl added	240 cc. N/30 HCl added	240 cc. N/30 HCl added	.....	.....	.....	.....	.....	.....
3/ 7/27.....	++	0	0	.....	.....	.....	.....	.....	.....
3/ 8/27.....	+++	++	0	.....	.....	.....	.....	.....	.....
3/ 9/27.....	++	+	0	.....	.....	.....	.....	.....	.....
3/10/27.....	0	0	++	.....	.....	.....	.....	.....	.....
3/11/27.....	0	0	++	.....	.....	.....	.....	.....	.....
3/15/27.....	N/30 HCl omitted	N/30 HCl omitted	N/30 HCl omitted	240 cc. N/10 HCl 8 Gm. calcium lactate added	.....	.....	.....	.....	.....
3/16/27.....	.....	.....	.....	+	.....	.....	.....	.....	.....
3/17/27.....	.....	.....	.....	++++	.....	.....	.....	.....	.....
3/18/27.....	0	0	0	++++	.....	.....	.....	.....	.....
3/19/27.....	0	0	+	.....	.....	.....	.....	.....	.....
3/22/27.....	0	+	0	.....	.....	.....	.....	.....	.....
3/28/27.....	45 cc. N/10 HCl added	45 cc. N/10 HCl added	45 cc. N/10 HCl added	45 cc. N/10 HCl added	.....	.....	.....	.....	.....
3/29/27.....	++	0	++	0	.....	.....	.....	.....	.....
3/30/27.....	0	0	+	.....	.....	.....	.....	.....	.....
4/ 1/27.....	++	++	+	.....	.....	.....	.....	.....	.....
4/ 2/27.....	0	0	0	++++	.....	.....	.....	.....	.....
4/ 3/27.....	+	0	0	++	.....	.....	.....	.....	.....
4/ 4/27.....	++	++	+	++++	.....	.....	.....	.....	.....
4/ 5/27.....	++	0	0	++++	.....	.....	.....	.....	.....
4/ 5/27.....	N/10 HCl omitted	N/10 HCl omitted	N/10 HCl omitted	N/10 HCl omitted	.....	.....	.....	.....	.....
4/ 6/27.....	0	.....	0	++++	.....	.....	.....	.....	.....
4/ 7/27.....	0	+	0	.....	.....	.....	.....	.....	.....
4/ 8/27.....	25 cc. N/10 HCl added	25 cc. N/10 HCl added	25 cc. N/10 HCl added	.....	100 cc. N/10 HCl added	100 cc. N/10 HCl added	.....	.....	.....
4/ 9/27.....	0	.....	0	+++	0	0	.....	.....	.....
4/11/27.....	0	0	0	0	++	0	.....	.....	.....
4/12/27.....	0	+	0	+++	0	0	.....	.....	.....
4/13/27.....	0	+	0	0	0	0	.....	.....	.....
4/14/27.....	0	+	0	0	0	.....	.....	.....	.....
4/15/27.....	N/10 HCl omitted	.....	N/10 HCl omitted	.....	.....	.....	.....	.....	.....
4/16/27.....	0	0	0	++++	240 cc. N/10 HCl added	.....	.....	.....	.....
4/17/27.....	0	0	0	+++	0	.....	.....	.....	.....
4/18/27.....	0	0	0	++++	++	.....	.....	.....	.....
4/19/27.....	0	0	0	++++	0	.....	.....	.....	.....
4/20/27.....	0	0	0	++++	+	.....	.....	.....	.....
4/21/27.....	45 cc. N/10 HCl added	45 cc. N/10 HCl added	45 cc. N/10 HCl added	Calcium lactate omitted	.....	.....	100 cc. N/10 HCl added	.....	.....
4/23/27.....	++++	+	0	0	0	.....	++	.....	.....
4/24/27.....	++	+	0	+	0	.....	+	100 cc. N/10 HCl added	.....
4/25/27.....	0	++	0	0	++	.....	++++	++	.....
4/26/27.....	0	++	0	+	0	.....	++	0	.....
4/27/27.....	0	++	+	0	+	.....	.....	0	.....
4/28/27.....	0	0	0	0	+	.....	+	0	.....
4/29/27.....	0	0	+	0	+	.....	.....	0	.....
4/30/27.....	0	0	0	0	++	.....	.....	++	.....
5/ 1/27.....	N/10 HCl omitted	N/10 HCl omitted	N/10 HCl omitted	.....	.....	.....	N/10 HCl omitted	.....	240 cc. N/10 HCl added
5/ 2/27.....	0	0	0	.....	+	.....	++	0	++
5/ 3/27.....	.....	.....	.....	.....	0	.....	.....	+	0
5/ 6/27.....	.....	.....	.....	.....	0	.....	+	0	++++
5/ 7/27.....	.....	.....	.....	.....	0	.....	.....	0	+++
5/ 8/27.....	.....	.....	.....	.....	++	.....	.....	0	++
5/ 9/27.....	.....	.....	.....	.....	.....	.....	.....	0	++
5/10/27.....	.....	.....	.....	.....	.....	.....	.....	0	+
5/11/27.....	.....	.....	.....	.....	.....	.....	.....	0	+++
5/12/27.....	.....	.....	.....	.....	.....	.....	.....	0	++
5/13/27.....	.....	.....	.....	.....	.....	.....	.....	0	++++
5/14/27.....	.....	.....	.....	.....	.....	.....	.....	0	++

* In the table, + = 1 to 2 casts; ++ = 3 to 10 casts; +++ = 11 to 20 casts; ++++ = more than 20 casts per 4 square centimeters.

casts; subsequently numerous casts of various kinds appeared, as shown in table 1. This infant was also given 8 Gm. of calcium lactate daily. The urine of two older children, 4 and 4½ years of age, who likewise received 240 cc. of tenth-normal hydrochloric acid with their milk, showed hyaline casts from twenty-four to forty-eight hours later.

The effect of a smaller quantity (100 cc.) of tenth-normal hydrochloric acid was next observed. This amount was added to the milk drunk by four older children, aged 4, 4, 4½ and 5 years. One of these did not show any changes in the urine after five days, at which time he left the institution. A few casts were noted at varying intervals in the urine of the three remaining children.

Four infants were next given a still smaller quantity (45 cc.) of tenth-normal hydrochloric acid with their milk. Casts in small numbers appeared in the urine of all these infants within from one to four days. In the case of the infant receiving calcium lactate in addition, casts appeared in larger numbers. When the acid was discontinued, the urine of all the infants cleared up rapidly with the exception of that of the last one; the calcium factor was responsible for the severe urinary changes in this infant, as will be shown subsequently.

As my co-workers and I were interested in determining the minimal amount of tenth-normal hydrochloric acid that could be added to milk without producing urinary changes, the quantity added was reduced to 25 cc. One of the three infants receiving this amount of acid showed merely from one to two casts on three occasions, and the others did not show any changes over periods of from seven to twelve days, respectively. The acid was then omitted from the diet, the urine was examined for five days, and all specimens became normal.

It was thought that possibly this development of casts on the addition of acid and their disappearance when the acid was discontinued might be merely a chance occurrence. In order to assure ourselves of the causal realtionship of the addition of acid, one more test was made Accordingly, the three infants were given 45 cc. tenth-normal hydrochloric acid daily. After two days in the case of two of the infants and six days in the third, the casts reappeared approximately to the same extent as during the period when this amount of acid had been given previously.

These results indicate that the changes in the urine associated with additions of tenth-normal hydrochloric acid to milk occur even when small quantities of acid are added.

We have noticed also that urine which shows casts on the addition of acid may become normal and remain so indefinitely even though the feeding of acid milk is continued; this leads us to infer that the kidney accustoms itself to the irritant. This was illustrated by E. R., who passed casts intermittently for two weeks when he received 240 cc. of

tenth-normal hydrochloric acid, but whose urine was normal for the seven following days. A similar occurrence was noted in C. W., who received 100 cc. of tenth-normal hydrochloric acid; the urine showed casts intermittently for six days and then became normal for a period of two weeks.

It was evidently of importance to note the hydrogen ion concentration of the urine under these conditions. It was found that the urine of infants receiving 45 cc. of tenth-normal hydrochloric acid and that of older children who received 100 cc. ranged uniformly between a p_H of 4 and 5 with an occasional specimen with a p_H of 6. In the case of E. R., who received 240 cc. of tenth-normal hydrochloric acid, the acidity was only slightly greater than when less acid was given. Apparently the urine cannot readily be acidified beyond a p_H of 4.

TABLE 2.—*Hydrogen Ion Concentration of Urine Following the Addition of Varying Amounts of Tenth-Normal Hydrochloric Acid to Milk*

Name	S. F.	B. K.	H. W.	A. W.	C. W.	E. R.	M. F.
4/16/27	...	...	...	...	...	240 cc. N/10 HCl added	...
4/21/27	45 cc. N/10 HCl added	45 cc. N/10 HCl added	45 cc. N/10 HCl added	100 cc. N/10 HCl added	...	...	...
4/22/27	4-5	4-5	6	4	5	4	...
4/23/27	5-6	4-5	5	4	4-5	4	...
4/24/27	4-5	5	5	4-5	100 cc. N/10 HCl added	4	...
4/25/27	5	5	5	5	5	4-5	...
4/26/27	4-5	5	5-6	5	4-5	4-5	...
4/27/27	5	5	5	4-5	4	5	...
4/28/27	5	4-5	5	5	4	5	...
4/29/27	5-6	5	5-6	5	4	5	...
5/ 1/27	...	...	...	...	...	...	240 cc. N/10 HCl added
5/ 9/27	...	5	...	...	6	...	4-5
5/10/27	...	5	...	...	6	...	4
5/11/27	...	6	...	...	5	...	4
5/12/27	...	5-6	...	...	4	...	4
5/13/27	...	5-6	...	...	4	...	5
5/14/27	...	5-6	...	...	4-5	...	4-5

THE EFFECT OF THE ADDITION OF CALCIUM LACTATE

That calcium may also act as an irritant of the kidneys was reported by Fuechtwanger and Lederer.[2] They observed granular and hyaline casts in the urine a few hours after giving calcium chloride; it disappeared when this medication was omitted. In the present study, the following instance shows the effect of the addition of calcium lactate: Infant M. M., aged 3½ months, suffering from typical tetany with severe convulsions, was given, in addition to 240 cc. of tenth-normal hydrochloric acid, 8 Gm. of calcium lactate daily. After twenty-four hours, his urine showed casts and red blood cells, which continued to be present for the following two days. A somewhat similar result was observed when only 45 cc. of this acid was added. When the acid was omitted, the urine still contained casts on eight different occasions, over a period

of thirteen days. When, however, the calcium lactate was discontinued, the urine became normal within fifteen hours and remained normal thereafter.

THE EFFECT OF THE ADDITION OF LACTIC ACID

A recent paper by Hottinger [6] gave interesting information concerning the excretion of some organic acid in infants. He found that even when large amounts of citric acid were fed, 95 per cent was consumed within the body, whereas when lactic acid was given in the form of buttermilk, a decidedly larger quantity of organic acid was excreted in the urine. The effect of lemon juice has been already discussed.

The effect of lactic acid was studied in two of the older children (4 and 5 years of age). To a quart of milk, two teaspoonfuls of lactic acid (U. S. P. 87 per cent) and 1 ounce of Karo syrup were added, according to the method of Marriott. In one instance, a few casts developed in the urine after three days; casts were not seen for the next six days, but they reappeared on the tenth, fourteenth and sixteenth day; from the seventeenth to the twenty-first day the urine was normal. In the case of the second child, a few casts were noted on the fourth and fifth day, but the urine remained normal thereafter.

The manifestations described in the urine resulting from the addition of acid or calcium to the milk raise the important question as to whether the casts result from an injury to the kidney or from a temporary irritation. It is generally believed that granular casts denote degenerative changes in the tubules of the kidney. In infants, however, conditions are not the same, for the kidney is far more sensitive to minor insults than in the case of the adult, as is illustrated by the well known pathologic changes which accompany such clinical conditions as acute intestinal intoxication, trauma in the new-born and "orthostatic albuminuria." The prompt disappearance of these changes when the source of the irritation has been removed is convincing evidence that the damage to the kidney has been slight and temporary. It would seem that this holds true for the fleeting pathologic conditions which we have occasioned. Seegal [7] recently published an experimental study of the effect on the kidney of feeding of tenth-normal hydrochloric acid. Although the rabbits passed casts and albumin throughout the experimental period, the only demonstrable pathologic change at necropsy was acute degeneration of the convoluted tubules. In those instances in which administration of acid had been stopped, it was found that the kidney had promptly returned to a normal condition. Similar results were obtained in experiments on dogs.

6. Hottinger, A.: Studien über Säuren- Basenhaushalt im kindlichen Organismus, Monatschr. f. Kinderh. **30**:497, 1925.

7. Seegal, B. C.: Chronic Acidosis in Animals: Relation to Kidney Pathologic Change, Arch. Int. Med. **39**:550 (April) 1927.

THE EFFECT OF SODIUM BICARBONATE

It seemed that it would also be interesting to note the effect of additions of an alkali to milk. With this in view, two infants were given 240 cc. of tenth-normal sodium bicarbonate with their milk; the taste of the milk was not appreciably altered, and the infants took their formulas well. Examinations carried out for six days failed to show any abnormal changes in the urine. These infants were next given 240 cc. of fifth-normal sodium bicarbonate with their milk; they likewise did not evince any urinary changes. These two infants, as well as the older children, were then given the same amount of a double normal sodium bicarbonate solution. Even this concentration of alkali did not prove irritating; examinations of urine were carried out daily for the following nine days. The reaction of the urine to litmus was alkaline, and the hydrogen ion

TABLE 3.—*Hydrogen Ion Concentration of Urine Following the Addition of Two Hundred and Forty Cubic Centimeters of Double Normal Sodium Bicarbonate to Milk*

Name	M. F.	O. W.	H. W.	S. F.
5/15/27	240 cc. double normal sodium bicarbonate	240 cc. double normal sodium bicarbonate	240 cc. double normal sodium bicarbonate	240 cc. double normal sodium bicarbonate
5/16/27	6.6	7.3	7.6	7.7
5/17/27	6.6	7.4	7.2	7.8
5/18/27	6.6	7.1	7.6	7.6
5/19/27	6.6	7.4	7.6	7.6
5/20/27	7.8	7.6	7.7	7.6
5/21/27	7.7	7.7	6.7*	6.7*
5/23/27	7.7	7.9	7.3	7.8
5/24/27	7.6	7.0	7.5	7.6
5/25/27	7.7	7.9	7.2	8.0

* Sodium bicarbonate omitted.

concentration ranged from 7.1 to 8 when the double normal solution was used. One exception should be noted in this regard, that of one of the older children who continued to pass acid urine with a p_H of 6.6 in spite of the fact that these large amounts of alkali were being added to the milk. In fact, the experience with sodium bicarbonate, an experience extending over a period of twenty-one days, has made it evident that large amounts of this alkali must be employed when one desires to bring about prompt and definite alkalinization of the urine. In this connection, attention may be called to the paper of Nassau [8] who gave some older children sodium bicarbonate in varying amounts from 7 to 10 Gm. in order to note the effect on "orthostatic albuminuria." Apart from its favorable influence on patients with this condition, it is worthy of note that this amount of alkali did not lead to signs of irritation of the kidney.

8. Nassau, E.: Ueber die Bedeutung der Reaktion des Harnes für das Auftreten der Statischen Albuminurie im Kindesalter, Ztschr. f. Kinderh. **33**:158, 1922.

THE EFFECT OF SODIUM HYDROXIDE

The effect of the addition of a stronger alkali, sodium hydroxide, was next studied. Two infants and two older children were each given 100 cc. of tenth-normal sodium hydroxide with their milk; their urine was examined for the next six days. It was found to be normal on each occasion. The reaction was acid to litmus, and the hydrogen ion concentration ranged from 5 to 7.2 with a preponderance on the acid side, much to our surprise. The amount of sodium hydroxide was now

TABLE 4.—*Hydrogen Ion Concentrations of Urine Following the Addition of Tenth-Normal Sodium Hydroxide in Varying Amounts to Milk*

Name	M. F.	C. W.	H. W.	S. F.
5/26/27	100 cc. tenth normal sodium hydroxide	100 cc. tenth normal sodium hydroxide	100 cc. tenth normal sodium hydroxide	100 cc. tenth normal sodium hydroxide
5/27/27	6.5	7.0	5.0	6.4
5/28/27	6.4	7.0	6.4	6.4
5/29/27	6.0	5.0	6.0	6.8
5/31/27	6.2	6.0	5.5	6.6
6/ 1/27	6.4	4.5	7.2	6.6
6/ 3/27	6.4	6.4	6.6	6.6
6/ 4/27	6.6	5.0	6.6	6.6
6/ 5/27	240 cc. tenth normal sodium hydroxide	240 cc. tenth normal sodium hydroxide	240 cc. tenth normal sodium hydroxide	240 cc. tenth normal sodium hydroxide
6/ 5/27	7.4	7.2	7.0	7.1
6/ 6/27	6.6	7.0	6.6	7.0
6/ 7/27	6.6	7.0	6.6	6.8
6/ 8/27	6.6	6.6	...	7.4
6/ 9/27	7.6	7.1	...	7.0
6/10/27	7.2	7.6	...	6.6
6/11/27	6.4	6.6	...	6.6
	240 cc. tenth normal sodium hydroxide; fat content of diet reduced	240 cc. tenth normal sodium hydroxide; fat content of diet reduced	...	240 cc. tenth normal sodium hydroxide; fat content of diet reduced
6/12/27	7.4	8.4	...	7.7
6/13/27	7.1	7.5	...	7.0
6/14/27	8.5	6.8	...	7.0
6/15/27	6.8	7.3	...	6.9
6/16/27	7.3	7.6	...	7.3
6/17/27	6.8	6.6	...	7.5
6/18/27	6.6	7.0	...	7.5

increased to 240 cc. Daily microscopic examinations for a period of fourteen days showed normal specimens of urine in each instance. The reaction was acid to litmus on many occasions and slightly alkaline less often, the hydrogen ion concentration ranging from 5 to 7.6 with a preponderance on the acid side. The stools of these children were alkaline and soapy.

It was apparent that the sodium hydroxide was being lost to the body, and that it was being excreted by way of the alimentary tract as evidenced by the alkaline and soapy stools. Our next step, therefore, was to prevent this elimination by the reduction of the fat intake of the food, which was accomplished by substituting skimmed milk and omitting

the butter from the diet. After this change, the reaction of the urine promptly became more alkaline, and the hydrogen ion concentrations rose to a range from 6.6 to 8.5 with a preponderance on the alkaline side.

CONCLUSIONS

1. Hydrochloric acid in small amounts, 45 cc. of a tenth-normal solution, when added to a liter of milk is followed by the appearance of casts and sometimes of red blood cells in the urine of infants. In older children, about 100 cc. of this dilute acid is required to bring about similar irritative manifestations.

2. When the tenth-normal hydrochloric acid is omitted from the diet, the pathologic urinary changes disappear in about forty-eight hours.

3. When 8 Gm. of calcium lactate was added to the milk, a similar result occurred.

4. Lactic acid (U. S. P. 87 per cent), when added in quantities of 8 cc. to a liter of milk, only occasionally produces casts in the urine.

5. Lemon juice in a concentration of 3 per cent does not lead to any evidences of irritation.

6. Sodium bicarbonate when given to infants and older children in large doses, 240 cc. of a double normal solution, the equivalent of 10.1 Gm. or 152 grains daily, does not have the same irritating effects on the urinary tract that acid does.

7. Sodium hydroxide, 240 cc. of a tenth-normal solution in quantities of 100 cc., does not produce the urinary changes noted when tenth-normal hydrochloric acid is added. The result is a slightly alkaline or slightly acid urine, which may account for the lack of irritative effects.

8. The addition of sodium hydroxide leads to the formation of the typical soapy stool.

9. When the fat content of the diet was reduced by skimming the milk, the addition of sodium hydroxide rendered the urine markedly alkaline.

CONGENITAL SYPHILIS

TREATMENT BY INTRAPERITONEAL INJECTION OF NEOARSPHENAMINE: REPORT OF TWO YEARS' WORK *

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Congenital syphilis is common enough to require the direction of particular attention toward the establishment of the best methods of treatment. Treatment of the unborn child through active treatment of the mother, while at times successful, is often unsatisfactory. The various forms of treatment of the child can be limited to the use of inunctions of mercury or mercury administered by mouth, potassium iodide, mixtures of bismuth or the arsenical compounds. While many satisfactory results have been obtained with mixtures of bismuth, it seems from present knowledge that the arsenical compounds will probably continue to give the most uniformly satisfactory results. Administration of the arsenical compounds to a child, however, is difficult. Intramuscular injections are often accompanied by the formation of abscesses, and the ideal procedure of giving intravenous injections is hampered by the small size of the available veins in the infant and the difficulty of injection.

Any method that will offer an easy means of administration, combining safety with rapidity of action, would be the procedure of choice. Rosenberg [1] was the first to report a case in which arsenic was injected intraperitoneally. Experiments were made on animals to obtain information concerning the formation of adhesions or other pathologic conditions of the peritoneum.

Early in 1924, experiments on animals were begun in our laboratory, and after four months the results were so satisfactory that they were reported by Sanford [2] while ten patients were receiving treatment. The method of administration by intraperitoneal injection has been in use

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* From the Department of Pediatrics, Rush Medical College of the University of Chicago, and the Pediatric Wards of the Presbyterian Hospital.

1. Rosenberg, William: The Intraperitoneal Administration of Neoarsphenamine, J. A. M. A. **82**:682 (March 1) 1924.

2. Sanford, Heyworth N.: Treatment with Intraperitoneal Injection of Neoarsphenamine, J. A. M. A. **85**:242 (July 25) 1925.

and under investigation in the Presbyterian Hospital for the past two years. The results have been so uniform and free from untoward effects that it has become a routine method of treatment.

The danger to the peritoneal contents was first studied by a series of experiments on animals;[3] this served as a basis for later clinical work.

EXPERIMENTS ON ANIMALS

The safety of intraperitoneal injections as regards life was well established in animals. In our series, fourteen rabbits, nine guinea-pigs and twenty-two dogs were injected from one to seven times, with no fatalities. There was some nausea in dogs after injection with arsphenamine dissolved in distilled water, but this disappeared entirely when a weak alkaline solution of 1 per cent sodium bicarbonate was used instead of water. Such an effect was never obtained in children, so the alkali was never used.

TABLE 1.—*Results of Injections into Various Parts of the Abdomen in Dogs*

Number of Injections	Position	Pathology
6	Right lower quadrant	None
6	Left lower quadrant	None
7	Upper left quadrant	3 Omentum adherent to self 2 Omentum adherent to parietal peritoneum 2 Omentum adherent to intestine
3	Upper right quadrant	1 Omentum adherent to intestine 2 Omentum adherent to mesentery

The work to make intraperitoneal injections safe as regards permanent changes in the cavity is of exceptional interest. All of the dogs that were given injections into the upper quadrants showed permanent changes. These changes may be classified as follows: adhesions of the omentum (1) to itself, (2) to the perietal peritoneum, (3) to the intestine and (4) to the mesentery. The results of injections into various parts of the abdomen in dogs is given in table 1.

It will be observed that a proper point of injection is essential. Two of the cases of adhesions between the intestine and the mesentery would have been serious in a mechanical way. Injections into the upper quadrants of dogs are therefore highly dangerous; injections into the lower quadrants, however, do not cause local changes and are free from danger.

METHOD

In the clinical cases here reported, the method of administration consisted of a sterile preparation of the abdomen similar to that used in giving any other intraperitoneal injection. The whole abdomen was washed with green soap and

3. The experimental work on animals was done by B. E. Noble and P. L. Heitmeyer.

water, then with alcohol. The ideal site for entrance of the needle is located in the middle of the sheath of the left rectus, slightly below the level of the umbilicus. This spot was chosen because experiments on animals showed that it is necessary to go below any possible limit of the omentum. It must be remembered that a site too far below the umbilicus will endanger the bladder. For this reason, as in many children the liver is at the level of the umbilicus on the right side, the middle of the sheath of the left rectus, slightly below the level of the umbilicus makes an ideal site for injections. This spot is painted with iodine and washed off with alcohol. The needle, preferably of small gage (18 gage), is inserted as for an ordinary intraperitoneal injection. The solution consists of 150 mg. of neoarsphenamine dissolved in 15 cc. of warm, sterile water. The child of average weight, 10 pounds (4.5 Kg.), should receive 50 mg. or 5 cc. of the solution. It should be emphasized that the needle must be pushed well through the peritoneum and into the peritoneal cavity. The substance is then injected as fast as the plunger will fall in the syringe, and sprayed over the peritoneal contents.

In only one instance in this course of treatments was there any reaction, and in that case the baby weighed only 5 pounds (2.3 Kg.) and received the full 50 mg. dose. A Herxheimer reaction of livid red patches, slight cyanosis and vomiting resulted. It disappeared spontaneously in ten minutes. Rapidity of injection and concentration of solution made little difference in effectiveness; it was found, however, that 5 cc. was an ideal amount.

The usual treatment consists of four injections at three day intervals followed by four injections at seven day intervals, the same dose being used each time. This is sufficient to clear whatever clinical pathologic condition due to syphilis exists, including the Wassermann reaction. The child is then turned over to the outpatient department for observation and returned to the hospital at the end of one month, when another Wassermann test is made and roentgenograms are taken.

COMMENT

Twenty-five patients were treated by the method described. Two were given neoarsphenamine intraperitoneally as provocative tests following anticomplementary and split Wassermann reactions. This reaction cleared up after one injection. Of the remaining twenty-three patients, sixteen, or 70 per cent, were pronounced clinically cured, with negative Wassermann reactions and negative roentgenograms. Four patients, or 17 per cent, showed distinct clinical improvement, but were removed from the hospital by their parents before treatment could be concluded. Three children, or 14 per cent, died during treatment; only one of these deaths could be attributed to the form of treatment. The observations made at autopsy are given in table 3

The last patient that died (case 6 in table 2) the tenth in order of treatment, is of interest. This child had a severe case of syphilis and

TABLE 2.—*Clinical Data on Authors' Patients*

No.	Age	Wassermann Reaction	Roentgen-Ray Examination	Symptoms	Reasons for Giving Intraperitoneal Injections	Number of Injections	Amount Mg.	Results
1	Birth	++++	Periostitis of femur	Vesicle on hands and feet; palpable spleen	Age	8	50	Disappearance of lesions; clearing up of roentgenogram and Wassermann reaction; gain in weight, 1 pound (0.5 Kg.)
2	3 wk.	++++	Extensive periostitis	Mottled skin; no gain in weight; palpable spleen	Age	8	50	Full use of arm; negative roentgenogram and Wassermann reaction; gain in weight, 2 pound 4 ounces (1.0 Kg.)
3	3 wk.	++++	Negative	Failure to gain; adenopathy; enlarged liver and spleen; coagulation time, 16 minutes; bleeding time, 26 minutes	Age	8	50	Gain in weight, 2 pounds (0.9 Kg.); negative roentgenogram and Wassermann reaction; bleeding time, 8 minutes; coagulation time, 10 minutes
4	4 wk.	++++	Periostitis of left femur	Pseudoparalysis of Parrot.....	Age	8	50	Full use of arm; negative roentgenograms and Wassermann reaction; gain in weight, 2 pounds 10 ounces (1.18 Kg.)
5	4 wk.	++++	Extensive periostitis	Bleeding from nose and umbilicus; generalized adenopathy; palpable spleen; coagulation time, 17 minutes; bleeding time 110 minutes	Age	6	50	At 7 weeks, gained 1 pound (0.5 Kg.); Wassermann reaction anticomplementary; bleeding time, 3 minutes; coagulation time, 5 minutes; at 8 weeks died of otitis media and intoxication; no adhesions or pathologic changes in the abdominal cavity
6	2 mo.	++++	Extensive periostitis	Scaly skin; ragades; saddle nose; loss of weight; adenopathy; enlarged liver and spleen	Age	7	50	At 3 weeks, gained 2 pounds 4 ounces (1.0 Kg.; at 7 weeks, was given injection in rectus muscle instead of peritoneal cavity; a sloughing ulcer developed that caused death
7	3 mo.	++++	Periostitis of long bones	Loss of weight; adenopathy; enlarged liver and spleen	Age	2	50	Gained 5 ounces (0.14 Kg.) during week; parents then removed child from hospital
8	3 mo.	+++	Negative	No symptoms; father and mother syphilitic	Age	3	50	Wassermann reaction cleared
9	3 mo.	++++	Periostitis of long bones	Acutely ill of bronchopneumonia; adenopathy; enlarged liver and spleen; bleeding time, 77 minutes; coagulation time, 4½ minutes	Age	2	50	In 48 hours, bleeding time was 5 minutes; coagulation time, 4 minutes; in 54 hours, child died of bronchopneumonia; autopsy revealed no adhesions or pathologic changes in the abdomen
10	3 mo.	++++	Periostitis	Snuffles; history of pemphigus; adenopathy; enlarged spleen	Age	4	50	Gained 10 ounces (0.28 Kg.) in 2 weeks; Parents then removed child from hospital
11	3 mo.	++++	Destructive changes	Maculopapular rash; adenopathy; enlarged liver and spleen	Age	3	50	Skin cleared; spleen not palpable; gained 10 ounces (0.28 Kg.) in 2 weeks; parents then removed child from hospital
12	3 mo.	++++	Periostitis	Adenopathy; enlarged liver and spleen	Age	8	50	Gained 2 pounds (0.9 Kg.); Wassermann reaction negative; roentgenogram showed clearing up of periostitis

13	3 mo.	++++	Extensive periostitis	Snuffles; adenopathy; enlarged spleen	Age	8	50	Lesions cleared up; Wassermann reaction negative
14	3 mo.	++++	Negative	No symptoms; parents actively syphilitic	Age	4	50	Wassermann reaction cleared; no symptoms; turned over to outpatient department
16	4 mo.	++++	Periostitis	Copper colored rash on body; palspleen	Small veins	8	50	Gained 2 pounds 6 ounces (1.17 Kg.); lesions cleared up; Wassermann reaction negative
16	4 mo.	++++	Periostitis of femur	Copper colored rash on body; palpable spleen	Small veins	8	50	Lesions cleared up; roentgenogram negative; Wassermann reaction negative
17	5 mo.	++++	Periostitis	Failure to gain; palpable spleen....	Small veins	8	50	Gained 2 pounds 3 ounces (0.98 Kg.); lesions cleared up; Wassermann reaction negative
18	5 mo.	++++	Periostitis	Circinate rash; palpable spleen.....	Veins small	1	50	Parents removed child from hospital
19	8 mo.	++++	Periostitis	Failure to gain; saddle nose; palpable spleen	Veins small	8	50	Gain in weight, 2 pounds (0.9 Kg.); negative Wassermann reaction; no change in roentgenogram
20	9 mo.	++++	Not typical of syphilis	Pseudoparalysis of Parrot; adenopathy; enlarged spleen	Veins small	7	50	Full use of arm; negative Wassermann reaction
21	11 mo.	*	None	Spleen just palpable; miscarriage history in mother	Veins small	1	50	Provocative Wassermann reaction negative in both antigens
22	14 mo.	++++	Negative	Condyloma; adenopathy; palpable liver and spleen	Veins small	8	75	Lesions cleared up; negative Wassermann reaction
23	2 yr.	++	Periostitis	Spasmophilia; blood calcium 7.7; six convulsions in 12 hours; no evidence of syphilis	Convulsions	6	75	Calcium given; no convulsions; blood calcium rose to 10.5; Wassermann reaction and roentgenogram negative
24	3 yr.	+++	Not characteristic	Copper colored rash on body; plantar ulcer; palpable spleen	Veins small	1	100	Parents removed child from hospital
25	2 yr.	++	Negative	Copper colored macules on body...	Veins small	2	100	Lesions cleared up; Wassermann reaction negative

* Cholesterinized antigen, +; acetone insoluble antigen, ++.

was in a bad condition. She was 2 months old and weighed 5 pounds, 4 ounces (2.4 Kg.). She received seven injections and gained 2 pounds, 4 ounces (1.0 Kg.). By mistake the eighth injection was given into the sheath of the rectus instead of into the abdomen. An abscess developed in three days, and the child died eight days later. Autopsy showed an abscess 4.5 cm. by 2.5 cm. in the muscular layer of the abdominal wall, containing thick, grayish-green pus. The transverse colon was adherent to the peritoneum by fibrinous adhesions under the abscess. There was a grayish-green, fibropurulent exudate in the leptomeninges of the entire surface of the brain. By cultural methods, *B. coli* and *Streptococcus viridans* were isolated from the spinal fluid and the abdominal abscess. Whether the poor condition of the child resulted in a lowered resistance is uncertain. It is clearly shown, however, that intraperitoneal injections of neoarsphenamine are not without danger if care is not taken in placing the needle in the abdomen.

TABLE 3.—*Autopsy Observations*

Number of Injections	Cause of Deaths	Pathologic Process in Abdomen
6	Alimentary intoxication	None
2	Bronchopneumonia	None
8	Acute meningitis, secondary to abdominal abscess	Abdominal abscess, transverse colon adherent to peritoneum

INDICATIONS FOR USE

The experience of two years has lead us to conclude that intraperitoneal injection of neoarsphenamine is a rational therapeutic method for treating those who have congenital syphilis. It is especially indicated in conditions in which rapidity of action is required, and the small veins of the child make intravenous injection impossible.

The rapidity of action is best shown in two cases of hemorrhagic syphilis in which this treatment was used. The first patient was a child, aged 4 weeks, who had hemorrhage from the cord, nose and anus, with a bleeding time of one hour and fifty minutes and a coagulation time of seventeen minutes. This child did not improve when given whole blood intraperitoneally and intramuscularly. The bleeding time continued to be one hour and twenty minutes, with a coagulation time of eleven minutes. Two hours after neoarsphenamine had been given intraperitoneally, the bleeding stopped, and the coagulation time fell to six minutes and the bleeding time to forty minutes. After another injection, the bleeding time fell to three minutes and coagulation time to five minutes.

The second case was one of hemorrhagic syphilis complicating lobar pneumonia. The coagulation time in this instance was four and a half minutes and the bleeding time seventy-seven minutes. After 40 cc. of

mother's whole blood was given, the coagulation time fell to four minutes and the bleeding time to forty-eight minutes; two hours after neoarsphenamine was given intraperitoneally, the coagulation time fell to four minutes and the bleeding time to five minutes.

SUMMARY

1. In twenty-five cases, covering two years' experience, two patients were given single injections of neoarsphenamine for diagnostic purposes. Of the other twenty-three patients, sixteen, or 70 per cent, were cured; four, or 17 per cent, were improved, and three, or 14 per cent, died. Of those who died, one patient, or 4 per cent, died as a result of the treatment.

2. Intraperitoneal injection of arsphenamine is not without danger if faulty technic is used.

3. Intraperitoneal injection of arsphenamine is indicated in the treatment of children who have congenital syphilis; rapidity of action is desired and the small veins make intravenous injection impossible.

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RESPIRATORY METABOLISM IN INFANCY AND IN CHILDHOOD

VII. ELIMINATION OF WATER THROUGH THE SKIN AND RESPIRATORY PASSAGES OF INFANTS *

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In a recent communication [1] it was shown that the relation between heat production and insensible perspiration was the same in children as in adults.[2] Under standard conditions of temperature, humidity and clothing, both adults and children lose an average of 24 per cent of their calories in vaporization of water through the skin and lungs. This proportion is maintained in sleep, during activity (insufficient to cause sweating), following the ingestion of food, in disease and with fever, providing the body temperature does not change during the observation.

If it were found that infants when exposed to similar conditions lost the same percentage of calories in vaporization as adults and children, the evidence would be adequate to establish a uniform relation between production of heat and loss of heat by vaporization (elimination of water) from early infancy to old age, despite the enormous differences in metabolic rates. The present investigation was undertaken in view of the rarity of such studies.[3]

Reviews of the literature on insensible perspiration and elimination [4] of water in adults [2] and in children [1] have recently appeared, and will not be presented in this paper.

* Received for publication, August 13, 1927.

* From the Department of Pediatrics, Cornell University Medical College and the New York Nursery and Child's Hospital.

1. Levine, S. Z., and Wilson, J. R.: Respiratory Metabolism in Infancy and in Childhood: IV. Elimination of Water through the Skin and Respiratory Passages of Children, Am. J. Dis. Child. **33**:204 (Feb.) 1927.

2. Soderstrom, G. F., and Du Bois, E. F.: Clinical Calorimetry: XXV. The Water Elimination through Skin and Respiratory Passages in Health and Disease, Arch. Int. Med. **19**:931 (May) 1917. Benedict, F. G., and Root, H. F.: Insensible Perspiration: Its Relation to Human Physiology and Pathology, Arch. Int. Med. **38**:1 (July) 1926.

3. Nieman, A.: Der respiratorische Gaswechsel im Säuglingsalter, Ergeb. d. inn. Med. u. Kinderh. **11**:32, 1913.

4. Although the terms insensible perspiration and elimination of water are used interchangeably, they are not identical. The former includes other gaseous emanations than water vapor, the chief component being carbon dioxide. Atmospheric oxygen is absorbed at the same time as carbon dioxide is exhaled. The difference or the weight of the carbon together with the water vapor constitutes the total loss in weight or insensible perspiration. The fraction ascribable to carbon is between 10 and 15 per cent, depending on the respiratory quotient.

METHODS

A description of the infant chamber and the modified "Benedict Universal Respiration Table" used to make simultaneous measurements of the production of heat and elimination of water in infants appeared in earlier papers of this series.[5] The accessory apparatus, including calorimeter bed, clothing and method for collecting urine, as well as the technic, methods of calculation and sources of error were also reviewed.[1-5] The infants were studied under the same standard conditions of humidity, temperature and clothing as the adults and children previously studied by Soderstrom and Du Bois [2] and by us.[1] The results are, therefore, strictly comparable. As heretofore, this paper includes only those observations which complied with the following requirements: The relative humidity of the air in the chamber changed less than 10 per cent during the observation, the range being between 35 and 60 per cent in all experiments. The change in the temperature of the air did not exceed 1 C. during the experiment, the range throughout being between 22 and 26 C. The rectal temperature changed less than 1 F. during the observation.

The relation between production of heat and elimination of water may be expressed in terms of calories produced and grams of water eliminated (Benedict and Root) or in terms of percentage of total calories vaporized (Soderstrom and Du Bois, Levine and Wilson).[6] For our purpose, the latter method is preferable as it affords the opportunity to compare directly the values found in infants with those previously found in adults and children.

ALCOHOL CHECKS

Observations on infants were controlled by frequent alcohol checks. The results for the year 1925-1926 appeared in the sixth paper of this series.[5] The apparatus measured the water produced by the alcohol flame with an average error of —0.5 per cent and a maximum error of —11.0 per cent on January 16. The figures for the following year are shown in table 1. The average error in the measurement of water was —2.7 per cent, the maximum error being —9.3 per cent on November 3.

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5. Levine, S. Z., and Wilson, J. R.: The Respiratory Metabolism in Infancy and in Childhood: I. Basal Metabolism in Children, Am. J. Dis. Child. **31**:323 (March) 1926. Wilson, J. R.; Levine, S. Z., and Rivkin, H.: The Respiratory Metabolism in Infancy and in Childhood: II. Ketosis and the Respiratory Exchange in Children, Am. J. Dis. Child. **31**:335 (March) 1926. Levine, S. Z.; Wilson, J. R.; Berliner, F., and Rivkin, H.: The Respiratory Metabolism in Infancy and in Childhood: VI. The Specific Dynamic Action of Foods in Normal Infants, Am. J. Dis. Child. **33**:722 (May) 1927.

6. At temperatures between 22 and 25 C., the vaporization of 1 gram of water requires 0.584 calories.

TABLE 1.—*Alcohol Checks for Experimental Year (1926-1927)*

Date	Minutes	Alcohol Gm.	Water			Carbon Dioxide			Oxygen			Respiratory Quotient (0.667)		Heat
			Theory Gm.	Found Gm.	Error per Cent	Theory Gm.	Found Gm.	Error per Cent	Theory Gm.	Found Gm.	Error per Cent	Found	Error	Theory Calories
10/4	56	....	....	5.22	.....	....	6.92	.....	....	7.63	.....	.....	.....	.....
	60	....	....	4.87	.....	....	7.31	.....	....	7.77	.....	.....	.....	.....
	Average	3.82	4.90	5.05	+3.1	7.30	7.12	—2.5	7.96	7.70	—3.2	0.672	+0.005	27.04
10/8	60	....	....	4.85	.....	....	7.26	.....	....	7.77	.....	.....	.....	....
	60	....	....	4.62	.....	....	7.13	.....	....	7.68	.....	.....	.....	....
	Average	3.82	4.90	4.74	—3.3	7.30	7.20	—1.4	7.96	7.73	—3.0	0.677	+0.010	27.04
11/3	60	....	....	4.06	.....	....	6.66	.....	....	7.26	.....	.....	.....	....
	60	....	....	4.14	.....	....	6.59	.....	....	7.20	.....	.....	.....	....
	Average	3.53	4.52	4.10	—9.3	6.74	6.63	—1.6	7.35	7.23	—1.6	0.666	—0.001	24.96
12/3	60	....	....	3.79	.....	....	5.46	.....	....	6.03	.....	.....	.....	....
	60	....	....	2.96	.....	....	4.76	.....	....	5.09	.....	.....	.....	....
	Average	2.70	3.46	3.37	—2.6	5.16	5.11	—1.0	5.63	5.66	+0.5	0.668	+0.001	19.11
1/10	45	....	....	....	.....	....	5.53	.....	....	6.02	.....	.....	.....	....
	56	....	....	....	.....	....	6.39	.....	....	6.96	.....	.....	.....	....
	* Average	3.09	....	....	.....	5.90	5.96	+1.1	6.43	6.49	+0.9	0.668	+0.001	21.84
2/2	60	....	....	....	.....	....	5.16	.....	....	5.77	.....	.....	.....	....
	60	....	....	....	.....	....	6.97	.....	....	7.51	.....	.....	.....	....
	* Average	3.27	....	....	.....	6.17	6.07	—1.7	6.74	6.64	—1.5	0.664	—0.003	23.14
2/29	60	....	....	2.53	.....	....	4.98	.....	....	5.48	.....	.....	.....	....
	35	....	....	1.75	.....	....	2.81	.....	....	2.94	.....	.....	.....	....
	Average	2.84	2.68	2.64	—1.3	3.99	3.90	—2.3	4.35	4.21	—3.2	0.673	+0.006	14.77
Total average			20.46	19.90	—2.7	42.56	41.99	—1.1	46.42	45.66	—1.6	0.670	+0.003	19.25

* The water produced by the alcohol flame was not determined in these experiments.

The checks were made with the bed and clothing in the chamber, and under the same conditions as observations with patients. The water vaporized by the alcohol flame in these checks was invariably less than the water output of the infants studied. In view of the careful technic and the close agreement in the alcohol checks, the average error in the results with subjects probably does not exceed 10 per cent.

RESULTS

Ten infants between 1 and 11 months old were studied in fifty-two calorimeter observations of from one and one-half to four hours' duration. All observations were made during sleep and from one to two

TABLE 2.—*Normal Infants*

Case	Date	Calories per Hour	Water Gm. per Hour	Calories Lost in Vaporization per Cent	Comment
1	1/13/26	14.1	6.4	26.4	Boy, 2-3 months old
	1/15/26	15.6	6.7	25.1	
	1/27/26	16.8	7.5	26.1	
	1/29/26	15.3	7.3	27.8	
	2/1 /26	15.2	6.9	26.4	
	2/3 /26	14.5	6.8	27.5	
	2/5 /26	15.6	7.3	27.4	
	Average			26.7	
2	4/16/26	13.8	6.6	28.0	Boy, 4 months old
	4/16/26	13.9	7.7	32.2	
	4/19/26	14.3	6.2	25.3	
	4/19/26	18.3	6.4	20.5	
	4/21/26	13.1	6.3	28.3	
	4/23/26	14.3	7.7	31.9	
	4/26/26	17.2	9.3	31.7	
	4/28/26	13.5	7.3	31.4	
	5/3 /26	14.9	7.9	31.0	
	Average			28.9	
6	11/8 /26	20.7	8.0	22.5	Boy, 10-11 months old
	11/8 /26	19.8	7.8	23.1	
	11/10/26	19.8	8.6	25.4	
	11/12/26	22.3	9.4	24.6	
	11/25/26	22.2	11.3	29.7	
	Average			25.1	
9	3/14/27	7.8	3.4	25.4	Boy, 1 month old
	Total average			26.5	

hours following small or moderate meals of casec, cream, glucose or dilutions of whole milk. The meals contained from 100 to 200 cc. of fluid.

The histories and calorimeter data of two normal infants (cases 1 and 2) were given in the sixth paper of this series.[5] The data for the remaining subjects will appear in subsequent communications.

The Water of Vaporization in Normal Infants.—Twenty-two observations were made on four well-nourished infants. A summary of the results appears in table 2.

The heat dissipated in vaporization of water by the normal infants averaged 26.5 per cent of the total caloric output, the range for indi-

vidual infants being between 25 and 29 per cent. These values parallel closely to those previously found in normal adults and in children. The average value in the latter subjects was 24 per cent, the extreme figures for individuals being 21 and 29 per cent.

The Water of Vaporization in Marantic Infants.—Six marantic infants were studied in thirty observations. The extent of their mal-

TABLE 3.—*Marantic Infants*

Case	Date	Calories per Hour	Water Gm. per Hour	Calories Lost in Vaporization per Cent	Comment
3	2/22/26	12.1	5.6	27.1	Boy, 5 months old
	2/24/26	11.7	4.8	23.9	
	2/26/26	13.8	5.3	22.5	
	2/27/26	14.1	6.0	24.9	
	Average			24.6	
4	4/11/26	10.1	4.7	26.9	Boy, 5 months old
5	10/18/26	16.4	5.8	20.6	Boy, 4-5 months old
	10/22/26	12.9	4.1	18.4	
	10/25/26	13.1	5.4	24.1	
	11/1 /26	11.4	5.6	28.5	
	11/2 /26	15.1	5.0	19.4	
	11/19/26	13.6	5.3	22.8	
	11/22/26	15.5	8.3	33.3	
	12/22/26	16.3	9.5	34.0	
	Average			25.1	
5	1/12/27	17.9	7.8	25.4	Same patient, 6-7 months old
	1/14/27	16.5	8.7	30.6	
	1/19/27	18.1	9.4	30.4	
	1/21/27	18.5	6.4	20.3	
	1/24/27	17.7	8.0	26.5	
	1/26/27	16.7	8.9	31.0	
	Average			27.4	
7	12/6 /26	7.3	2.8	22.8	Boy, 2 months old
8	12/22/26	15.4	8.6	32.4	Boy, 6-7 months old
	12/31/26	16.1	7.2	26.0	
	1/3 /27	17.0	7.7	26.3	
	1/5 /27	16.6	10.0	35.1	
	Average			30.0	
10	2/14/27	8.1	3.1	32.1	Girl, 3 months old
	2/16/27	9.7	3.9	23.3	
	2/21/27	8.5	3.4	23.9	
	2/22/27	9.4	3.2	20.1	
	2/23/27	9.1	3.5	22.7	
	2/25/27	10.2	4.4	24.9	
	Average			22.7	
	Total average			25.6	

nutrition varied from moderate to extreme severity, their body weights ranging from 20 to 64 per cent below normal standards for their respective ages.[7] One subject (case 5) was studied at intervals over a period of three months. During this time, his weight increased steadily from minus 47 to minus 20 per cent of the standards for the two age periods. The results are summarized in table 3.

7. Woodbury, R. M.: Statures and Weights of Children under Six Years of Age, Children's Bureau Publication, 87.

The heat lost in vaporization by the marantic infants averaged 25.6 per cent, the extreme figures for individual infants being 23 and 30 per cent. Despite the elevated basal metabolism (per actual weight and surface area) associated with marasmus, these subjects gave values which corresponded closely to the figures obtained in the well-nourished infants with normal basal metabolic rates. The trivial discrepancies are well within the experimental error of the method.

There is further evidence in favor of the view that under the standard conditions before enumerated the height of metabolism does not influence the proportion of calories removed in vaporization. Two infants (cases 4 and 5, second series of observations) whose basal metabolic rates were elevated 77 and 16 per cent above normal standards, respectively,[8] dissipated identical fractions of the total caloric output in vaporization, namely, 27 per cent (table 3).

COMMENT

The evidence available at the present time is sufficient to establish a strikingly uniform relationship between production of heat and elimination of water from early infancy to old age, in health and in many diseases. All the data were obtained under similar standard conditions of body and environmental temperature, humidity, clothing and activity. The technic was rigidly controlled throughout, and the average error of the method probably does not exceed 10 per cent. The figures obtained in all subjects are, therefore, strictly comparable.

Despite the enormous differences in metabolic rates in the age periods from infancy to adult life, apparently a definite and unvarying fraction of the total amount of heat produced by all subjects was removed from the body in vaporization of water through the skin and lungs (average of 26 per cent). There is no evidence at present to suggest that this proportion is maintained under conditions which induce active, visible sweating.

Benedict and Root [2] have recently made use of this close relationship in a practical way by constructing a straight line curve as a method for predicting the probable metabolism of adults from the measured insensible perspiration.[4] As it is particularly difficult and time-consuming to measure the metabolism of infants and children, a similar method for estimating the metabolism of these subjects would be extremely desirable. The present, earlier and additional data obtained with the use of a delicate balance under carefully controlled conditions will be incorporated in a paper which will appear shortly. Evidence will

8. Benedict, F. G., and Talbot, F. B.: Metabolism and Growth from Birth to Puberty, Pub. 302, Carnegie Inst. Washington, 1921.

be presented to show that the time-saving method suggested by Benedict and Root for predicting metabolism in adults is reliable for clinical purposes in infants and in children.

SUMMARY AND CONCLUSIONS

The elimination of water through the skin and respiratory tract of four normal and six marantic infants was measured in fifty-three calorimeter observations of from one and one-half to four hours' duration. The temperature and relative humidity in all experiments ranged between 22 and 26 C. and 35 and 60 per cent, respectively. The previous work with adults and children was done under identical experimental conditions. In view of the rigidly controlled technic, the experimental error probably does not exceed 10 per cent.

Under these conditions, the normal and marantic infants lost an average of 27 and 26 per cent of the total amount of heat produced in vaporization, respectively. The extreme average figures for both groups were 23 and 30 per cent. Only occasionally did the figures for individual observations exceed this range.

The close relation between insensible perspiration and metabolism previously found to exist in adults and children may be extended to infants, despite the notable differences in metabolic rates. It may be stated that under ordinary conditions the human subject from early infancy to old age dissipates approximately 25 per cent of the total amount of heat produced in vaporization of water through the skin and lungs, and the remaining 75 per cent by way of radiation and conduction.

ACUTE INTUSSUSCEPTION

MANIPULATIVE REDUCTION UNDER FLUOROSCOPIC CONTROL *

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BERWYN, ILL.

While examining a patient with acute intussusception under the fluoroscope several months ago, I noticed that the invading mass of bowel was pushed backward by the column of barium from its original position at the sigmoid flexure to a new one in the transverse colon, in which it was found half an hour later when the abdomen was opened for the purpose of carrying out the usual operative treatment in such cases. The patient made a successful recovery, but three months later the mother appeared at the office with the child and announced that she was certain that there was a recurrence of the old trouble. Her observation was confirmed by the finding of a movable tumor mass in the upper right quadrant of the abdomen and an unmistakable blocking of the column of barium distal to the hepatic flexure. As it met the invading ileum, the column of barium presented the characteristic features, which I have described in a previous article.[1] Extreme reluctance to perform a second abdominal operation on a child 6 months of age prompted me to attempt to reduce the intussusception by a barium enema and manipulation. This [2] was successfully done, and the case was reported at the Illinois State Medical Convention, May 30, 1927 (fig. 1).

I do not claim priority for this method, as Retan [3] of Syracuse, N. Y., reported two successful cases in the May number of the *American Journal of Diseases of Children,* which appeared a few days before my first case was presented, and which I believe are the first on record. Manipulative methods of reduction are not new, and while they are not looked on with favor in America, a great many patients have been successfully treated in Europe and Australia particularly by enemas of physiologic sodium chloride solution.[4]

The advantages of an opaque enema with fluoroscopic control of the manipulations should be obvious. In the first place, the opaque medium

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* Received for publication, July 30, 1927.

1. Stephens, V. R.: Acute Intussusception Without Pain, J. A. M. A. **88**:475 (Feb. 12) 1927.

2. Stephens, V. R.: Use of the Fluoroscope in Surgical Manipulations, Read at State Medical Convention, Moline, Ill., May 30, 1927. To be published.

3. Retan, G. M.: Nonoperative Treatment of Intussusception, Am. J. Dis. Child. **33**:765 (May) 1927.

4. Hipsley: Intussuception and Its Treatment by Hydrostatic Pressure, M. J. Australia **2**:201 (Aug. 14) 1926.

confirms the diagnosis which, in some cases, is more or less obscure. I recently operated on a patient in whom a tumor mass was not demonstrable, and there was no blood in the stool. The diagnosis was based solely on the fluoroscopic picture.[5] There was some evidence of shock, and the child had intermittent colic, but that is not sufficient evidence for a positive diagnosis. In the second place, visual control of the amount

Fig. 1.—Acute ileocecal intussusception with the apex in the proximal transverse colon outlined by contact with a barium enema. Reduction was accomplished by manipulation with the aid of the opaque medium.

of enema admitted into the bowel is obtained, and dangerous distention can be avoided. Finally, there need be no doubt about the completeness of the reduction in most cases.

5. Stephens, V. R.: Fluoroscopic Findings in Acute Ileocecal Intussusception, Surg. Gynec. Obst. To be published.

METHOD

An anesthetic is not required, as it is not necessary to procure enough distention to cause pain. If the child is frightened or stubborn, fill the colon with barium as far as the intussusception, using a large colon tube; hold it in position with the fingers to prevent leakage, and wait until the child becomes quiet. It is not advisable to allow any barium to escape, as it blurs the field of observation

Fig. 2.—Barium filled colon after completing the reduction. A generous sprinkling of barium is seen in the terminal ilium and the edema of the ileocecal region is shown by the decreased caliber at that point.

and tends to increase the invagination. Admit only a small amount at a time, work slowly and manipulate gently at the point at which the enema meets the intussusception. If there is much gas which obscures the outline of the head of the intussusception, allow the first enema to escape with the gas, keeping the child in the horizontal position, and refill. I found this necessary once when the presenting surface of the barium column was not typical in outline, in order to get

a good roentgenogram for demonstration. Do not depend on the barium pressure to reduce the intussusception, but admit just enough to keep the colon well filled to the point at which reduction has been accomplished. Be sure that the caput coli is filled. A generous trickle into the ileum is important evidence of complete reduction. A relaxed or incompetent ileocecal valve may be an etiologic factor in this type of invagination of the bowel. If in doubt concerning the completeness of the reduction, open the abdomen at once. In the open abdomen, it is a simpler procedure to reduce a mass that extends no farther than the hepatic flexure than one which has invaded the sigmoid and produced a partial volvulus of that flexure. I once experienced the latter condition in an open operation for ileocecal intussusception. If only partial success is attained, the procedure is well worth while if carried out carefully. This method is similar to that of Retan in essential points but differs in some details. I depend on one hand to do the manipulating and on the other to control the flow of barium in the tube so as to fill quickly the space vacated by the manipulation. I keep one hand continuously on the colon tube to hold it in place, as its expulsion causes delay and annoyance.

REPORT OF CASE

J. S., a boy, aged 2 years and 3 months, a well nourished, even tempered child with a history of normal birth and feeding had had no previous illness of note with the exception of a mild case of measles a year previously. On the evening of July 7, 1927, he ate a hearty dinner and appeared happy, but immediately after the meal he complained of stomachache and vomited violently. The mother said that he looked pale after he had vomited, but that his normal color returned in a few minutes. In fifteen minutes, the pain reappeared, with vomiting followed by pallor and relaxation. I arrived an hour later, and found the child pallid, moist and relaxed. The temperature was 98 F. by rectum; the pulse rate was 120 and the pulse was weak. Blood was not noticed following an enema given by the mother a few hours before. Physical examination revealed only a flaccid abdomen and an indefinite movable tumor mass over the region of the right kidney. The advice to make a fluoroscopic examination was accepted, and the observations are shown in the accompanying roentgenogram (fig. 2). The intussusception was reduced by the method previously described. No ill effect was noted. The following day the child played outdoors as usual.

CONCLUSIONS

Manipulative reduction should be attempted in early and moderately advanced cases. The degree of severity does not depend on the time element alone and the resultant opportunity for agglutination of surfaces, but on the extent of the process and the firmness of the incarceration and consequent interference with the circulation. After operating on five patients in the usual manner, with one death on the seventh day from an unknown cause, and after reducing two cases by manipulation under fluoroscopic control with perfect results, I recommend unhesitatingly the latter method in properly selected cases. If there is failure to reduce, no harm has been done if the procedure has been properly carried out, and appropriate surgical treatment can be instituted immediately.

ACUTE OSTEOMYELITIS OF THE SUPERIOR MAXILLA IN YOUNG INFANTS*

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I wish to report two cases of an unusual disease occurring in newborn infants—a condition characterized by a definite clinical syndrome, which presents the following picture: A newly born or a young infant suddenly becomes ill with fever and prostration and shows a swelling and redness about the tissues of one eye; this is followed or is accompanied by a purulent unilateral nasal discharge, by swelling and softening of the alveolar border of the superior maxilla on the affected side and by pointing and the formation of abscesses on the hard palate, the alveolar border and often on the face between the eye and the nose. Sequestrums and frequently small teeth are discharged from these sinuses.

REPORT OF CASES

CASE 1.—Doris C., born April 2, 1922, was the first child of healthy parents. The birth was normal; she was breast-fed exclusively for fourteen days, and following that received supplementary bottles. While still in the obstetrical hospital, she developed impetigo neonatorum. When I first saw her at the age of 18 days, most of the lesions on the skin had healed except a few in the left axilla. On April 23, when the child was more than 3 weeks old, the mother noticed that the left eye was reddened and that the tissues about it looked puffy. The child was seen by a physician who thought that the condition was a conjunctivitis and prescribed drops of a mild silver solution in the eye. The ocular condition seemed to improve a little, but that evening redness was noted about the left side of the nose. On April 28, pus was noted in the mouth, and the child was brought to me. The general condition was excellent. Fever was not present. On the left ankle was a small pustule, and over the occiput there were three lesions of impetigo. The left cheek was markedly swollen in the infra-orbital region. The area between the eye and the nose was red, hot, shiny and tender. The eye could be opened, and the conjunctiva and cornea were seen to be normal. Movements of the eyeball were unrestricted. The pupils were normal. A slight exophthalmos was noticed. Pressure over the swelling of the cheek caused exudation of pus from the left nostril and also from a small opening in the outer sides of the left superior alveolar process about where the canine tooth should be. When a probe was passed into this sinus bare bone was felt, and the probe could be introduced to a distance of about 2 cm. The end of the probe could not be detected by palpating the cheek. It appeared to be in a bony cavity.

On the following day, April 29, the child's condition appeared to be about the same. She was seen and examined by Dr. Harold Hays. Transillumination with a light in the mouth failed to show any difference between the two sides. When the light was placed over the antrum externally and the roof of

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the mouth was inspected, the left side was seen to be dark. There was a profuse purulent nasal discharge. Intranasal illumination failed to show the opening of the sinus. On May 2, the general condition was still good, but considerable pus exuded from the sinus and the nose. The swelling of the cheek and eye had diminished slightly. The small pustule on the left ankle was incised. The impetigo had improved. Culture of the pus from the sinus showed a pure growth of *Staphylococcus pyogenes-aureus.* The sinus was probed daily in order to keep it wide open, and on one occasion two small teeth were discharged with the pus.

On May 6, roentgenograms showed clouding over the region of the left antrum; the child looked worse, and the reddened area over the side of the nose seemed pointing. This swelling was incised by Dr. Hays and a small rubber tube inserted, through which the cavity in the maxilla was irrigated for the next three weeks. The condition gradually improved. When seen on July 5, at the age of 3 months, the child weighed 9 pounds and 14 ounces (4.4 Kg.). There was little purulent discharge from the mouth, but several small sequestrums of bone were removed. Some thickness was still present on the left side of the nose, but the incision there had completely healed. On August 4, at the age of 4 months, the child weighed 12 pounds and 1 ounce (5.4 Kg.). Pus still drained from both the mouth and the nose. On September 6, the weight was 14 pounds and 4 ounces (6.5 Kg.). The scar on the face had healed well. There was a small mass of granulation tissue at the site of the fistula in the mouth, but pus was no longer present. On October 9, the weight was 16 pounds and 7 ounces (7.5 Kg.). The mouth or the nose was no longer discharging. On March 9, 1923, at the age of 11 months, the infant weighed 19 pounds and 12 ounces (8.9 Kg.). The two upper and two lower central incisors were present. Two months later, two more upper incisors had appeared. On Nov. 8, 1924, the teeth had erupted properly, except those in the left upper posterior gum. At the site of the left upper canine was an irregular point in place of the tooth. On Oct. 23, 1925, at the age of 3½ years, the last left upper molar was absent; the first molar and the canine were merely stumps. In May, 1926, the child had a mild attack of scarlet fever. In November, 1926, she was in excellent condition. The nasal scar had almost disappeared. During the previous summer, one of the irregular upper teeth apparently had fallen out. The lower teeth were normal. The five right upper teeth were normal. The two left upper incisors were normal; in addition, there was an imperfect bicuspid, the two molars being missing. Roentgenographic examination showed slight relative clouding of the left antrum. There were no defects of the bones.

Case 2.—I first saw Thelma O. on Oct. 8, 1925, when she was 10 weeks old. The child had been normally delivered; she was breast-fed and had not suffered any previous illness, having been gaining well since birth. On October 4, she seemed irritable and cried a great deal. She was seen by a physician who noted a temperature of 101.5 F. but could find nothing to explain the fever. The next day the temperature was higher, and the mother noticed a swelling above the margin of the upper right gum. A surgeon was consulted, who incised the swelling and obtained pus. On the following day, October 7, the temperature rose to 104 and another swelling appeared on the roof of the mouth. This ruptured spontaneously. Soon the inflammation spread to the eye, and a purulent right-sided nasal discharge appeared. When I first saw the child, she appeared extremely ill, toxic and prostrated. She was admitted to Mount Sinai Hospital for study and treatment. She was pale.

The tissue about the right eye was markedly edematous and red, and the eye was closed. The eyeball moved well when the lids were retracted. There was distinct exophthalmos. The pupils reacted normally. The skin over the upper part of the right cheek was reddened and swollen. At the right upper alveolar border, where the canine tooth should be, there was a sinus leading to bare bone. In the midline on the hard palate, a similar sinus was present. There was a profuse purulent discharge from the right nostril. Examination otherwise gave negative results, as shown in the accompanying illustration.

On account of the septic appearance of the child, a blood culture was made which showed one colony of *Staphylococcus aureus* per cubic centimeter. A culture of pus from the sinus showed the same organism. Roentgenograms were unsatisfactory, owing to the restlessness of the child. She continued to have a high temperature for several days. Treatment consisted of frequent cleansing of the eye and dilatation of the oral sinuses to prevent their closing.

Two days after admission to the hospital, two new sinuses appeared, one on the right side of the hard palate near the posterior border and the other at the juncture of the lip and the alveolar border. These and the two sinuses previously described discharged pus profusely. When the sinuses were probed, small sequestrums were discharged, but no teeth. The temperature gradually

Marked swelling about the periorbital tissues in case 2.

came down to normal in the course of a week. The child took its feedings well; the general condition improved, and the orbital swelling completely disappeared. The sinuses remained open for about a week, with the exception of the one on the alveolar border, which was still open when the child left the hospital after a stay of ten days. The child made an uneventful recovery, and the last sinus closed after a few weeks.

COMMENT

In looking over the literature, I found a number of identical cases cited by foreigners, but curiously enough I cannot find a description of the disease in American journals. In 1924, Kelly [1] reported a case under the title "The So-Called 'Emypema of the Antrum of Highmore in Infants' (Osteomyelitis of the Superior Maxilla)." His case is an exact counterpart of those that I have described. He was able to collect reports of seventeen cases, which he has summarized in his paper. His

1. Kelly, A. B.: The So-Called "Empyema of the Antrum of Highmore in Infants" (Osteomyelitis of the Superior Maxilla), Edinburgh M. J. **16**:302, 1904.

case is the eighteenth on record. I have been able to find references to five other cases.[2] The condition therefore must be rare. The only reference to it that I found in the American literature was in a paper by Mayer,[3] in which he detailed the case of a child, aged 2 years, who had maxillary sinusitis. In the discussion of this case, he referred to a number of the cases quoted by Kelly.

The disease presents a clear-cut, definite picture, one case report being almost identical with the other. All the cases occur in young infants, ranging in age from 2 or 3 weeks to 9 months. The children are apparently in good health when they are suddenly attacked by an infection localized in the region of the superior maxilla. The first sign of disease is, as a rule, redness and swelling of the lids and periorbital tissues of one eye. The lids become puffy, and a purulent conjunctivitis may be present. The orbital swelling may increase to such an extent as to give rise to exophthalmos, as shown in the accompanying illustration. In the majority of cases this inflammation goes on to localization below the inner canthus of the eye, in which an abscess may form and rupture, leaving a fistula discharging on the face. Coincidentally with this, a thick purulent nasal discharge appears on the affected side. When the mouth is inspected, swellings are seen along the upper gum, usually about where the canine tooth should be, and also anywhere on the hard palate. These swellings either rupture spontaneously or are operated on. They discharge creamy pus mingled with particles of bone. The discharge of unerupted teeth from these sinuses is characteristic. When the alveolar sinuses are probed, the instrument enters a large cavity, on the walls of which bare bone is encountered. All of this occurs within a few days, during which time the infant appears very ill. The temperature is irregular, convulsions frequently are present, and there is marked anorexia and difficulty in nursing, due to the pus in the nostril. The outcome is either healing with or without the persistence of discharging sinuses or the development of secondary purulent foci; or else the infant dies as a result of the virulence of the onset before secondary foci have time to develop.

Though the clinical picture is sharply defined, as may be seen from the foregoing description, much confusion exists in the consideration of its pathogenesis. Many of the cases have been described as infections

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2. Canestro, C.: Entzündung der Highmorshöhle bei Neugeborenen, Arch. f. Laryngol. u. Rhinol. **25**:492, 1911. Hughes: Empyema of the Antrum in an Infant Three Weeks Old, Brit. J. Dis. Child. **18**:89, 1921. Castex, A.: Sinusite grave chez un nouveau-né, Rev. de stomatol., 1901, p. 218. Muggia, A.: Sulla sinusite purulenta mascellare dei neonati, Pediatria, 1909, p. 38. Weissmann, E., and Fiocre, L.: Le sinus et les sinusites maxillaires chez les nouveau-nés, Ann. d. mal. de l'oreille, du larynx **32**:225, 1906.

3. Mayer, Emil: Sinusitis in Young Children, M. Rec. **60**:210, 1901.

of the antrum of Highmore, others as osteomyelitis and others as phlegmons of the orbit. It is now known, chiefly through the work of Onodi,[4] that even in the new-born infant the maxillary sinus is present. It has the following dimensions: dorsoventral diameter, from 7 to 10 mm.; mediolateral, from 2 to 3 mm., and superior-inferior, from 3 to 4 mm. One can therefore conceive of a sinusitis, even in a new-born child. The clinical picture, however, does not really resemble antral disease and corresponds much more closely to an inflammation of the superior maxilla such as one occasionally sees following trauma in adults. Schmiegelow[5] was the first to show that these cases were in reality cases of osteomyelitis. As Kelly pointed out, the whole superior maxilla is involved, and evidence of its disease is presented by each of the bony surfaces—the orbital, nasal, facial and palatal. Moreover, when a probe is passed into one of the alveolar sinuses, it comes at once into a large cavity. This has been incorrectly supposed to be the antrum, though the true antral dimensions, as already stated, show that the sinus itself is a tiny structure. The cavity into which the probe enters is not the antrum but one of the dental sacs. The discharge of unerupted teeth in almost all the cases described still further implicates the maxilla itself.

Inflammation of the tooth-buds in the course of sepsis or erysipelas in the new-born has been described by Klementowsky, Swoboda and Zarfl.[6] The last author made a detailed histologic study of such cases and showed that a gangrenous process may arise in the undeveloped teeth. Zarfl called the disease "Sequestrierende Zahnkeimentzündung." In his papers he described two cases in young infants under the heading of "Osteomyelitic Form," in which the description fits the cases I have been describing. Both of these children died, and postmortem examination showed a histologic picture similar to the cases occurring in the course of sepsis. Finkelstein[7] and von Reuss[8] seem to have believed that osteomyelitis of the maxilla may begin about the tooth-buds.

The evidence, therefore, all seems to point toward the fact that this condition is a disease of the bone itself and not one of the maxillary antrum.

The immediate cause of this inflammation is different in the various cases. Some have followed delivery with forceps, with injury of the parts; some have been face presentations in which trauma has played a

4. Onodi, A.: Die Nebenhöhlen der Nase beim Kinde, Wurzburg, 1911.

5. Schmiegelow, E.: Ueber akute Osteomyelitis des Oberkiefers, Arch. f. Laryngol. **5**:115, 1896.

6. Zarfl, M.: Sequestrierende Zahnkeimentzündung im frühestem Kindesalter, Ztschr. f. Kinderh. **25**:266, 1920.

7. Finkelstein, H.: Saüglingskrankheiten, Berlin, 1921.

8. Von Reuss, A.: Die Krankheiten des Neugeborenen, Berlin, 1914.

rôle; some have apparently followed infection in the mother. The question of intrapartum intranasal infection has been mentioned, but this seems unlikely. In one of my cases the infant was apparently in excellent health, and the labor had been normal. In the other there was a furuncle on the ankle, and impetigo neonatorum had been present for two weeks. The factor of trauma did not appear to play a rôle. Both cases showed *Staphylococcous aureus* in the pus, and one showed the organism present in small numbers in the blood. The last fact speaks in favor of the osteomyelitic theory, as positive blood cultures are so frequently found in acute disease of the bones.

Contrary to what one would expect, the prognosis is not unfavorable. So extensive a process in such a young infant would appear almost surely fatal. However, in the twenty-four cases reported, six infants died and eighteen recovered.

The discharge of teeth, so characteristic of this disease, leaves the child without teeth over the affected area. In my first case, a white bony mass could be seen in the region of the first molar tooth two years after the illness. This was not disturbed, but was discharged when the child was 4½ years old and was evidently a deformed molar tooth.

The treatment consists of keeping the fistula open and of removing sequestrums as they appear. If the process points on the face, a small incision may be made without fear of leaving a large permanent scar. As foci point on the palate and gum, they should be opened. The eye must be protected with compresses and frequently cleansed. My second patient recovered after only the simplest surgical treatment and in spite of the positive blood culture and the high temperature.

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Case Reports

DERMATOMYOSITIS

REPORT OF A CASE WITH NECROPSY *

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AND
LEWIS A. KOCH, M.D.
OMAHA

Dermatomyositis is interesting because of its rarity and the striking resemblance in the clinical picture that all cases present. Steiner [1] defines it as an "acute, subacute or chronic disease of unknown origin, characterized generally by a gradual onset with vague and indefinite prodromata followed by edema, dermatitis and multiple muscle inflammation." Friedman [2] gives a complete presentation of the condition. To date, about fifty-five cases have been recorded. The following case was typical and presented excellent opportunities for study.

REPORT OF CASE

History.—A white boy, aged 5, was admitted to the University Hospital, Dec. 31, 1926, because of painful, swollen, contracted joints and generalized tenderness and weakness of the muscles. He also complained of edema and an erythematous rash about the face, chest and extremities and an associated, irregular, intermittent fever.

The patient had been born four weeks before term. His first illness had occurred in September, 1925, when he had been confined to bed for one week with diarrhea, fever and convulsions. Recovery seemed to be complete except for noticeable weakness of the muscles. In January, 1926, after he failed to gain strength, the mother noted swelling of the wrists and ankles. He was then unable to regain his feet when playing on the floor and complained of tenderness when lifted by the arms. In March, 1926, swelling and redness were noted about the eyes. He was taken to the family physician, who found pus in the urine and started treatment for pyelitis and rheumatism. No improvement followed and in August, 1926, the tonsils were removed. He was then treated with cod liver oil and urinary antiseptics until admission to the hospital.

The family history was negative.

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* Received for publication, Aug. 4, 1927.

* From the Department of Pediatrics, University of Nebraska College of Medicine.

1. Steiner, W. R.: Dermatomyositis, with Report of Two Cases, J. A. M. A. **78**:271 (Jan. 28) 1922.

2. Friedman, E. D.: Dermatomyositis, M. J. & Record **123**:382 (March 17) 1926.

Physical Examination.—On admission to the hospital, the boy was poorly nourished. He lay in bed in a dorsal fixed position of moderate flexion (fig. 1). The skin about the eyes and forehead was edematous and would not pit on pressure. This gave a peculiar masklike appearance to the face. Erythema was present over the edematous area. The chest was slightly edematous and presented many petechiae (fig. 2). About the axillae were several small crusted ulcerations

Fig. 1.—Characteristic posture of patient, showing flexion deformities of extremities.

Fig 2.—Edema about face and pitlike ulcers of skin.

and many small, bluish, circular scars apparently resulting from previous ulcers. The skin over the abdomen was slightly thickened but relatively clear. The upper extremities were emaciated and were held in natural flexion over the chest. A bronzing of the skin was noted over both arms and extended down to the dorsum of the hands. A small draining furuncle was present over each olecranon process. Flexion or extension of all joints was limited by muscle contractures, and passive motion was extremely painful. Both biceps were contracted, resulting

in partial flexion deformities of the elbows. The left wrist was swollen and tender and was held in extension by the contracted extensor muscles of the hand. The lower extremities did not present the bronzed condition noted on the arms, but numerous ulcerations and scars were present about the knees and feet. The ankles also presented the thickened edematous condition of the skin. Both hips and knees were moderately flexed. All other organs were normal. Results of neurologic examination were negative.

Laboratory Observations.—The urine showed decreased volume, constant pyuria and intermittent traces of albumin and sugar. Cultures of *B. coli* and *Staphylococcus aureus* were obtained from the urine. A leukocytosis was present, which ranged from 9,000 to 13,500 with from 70 to 80 per cent polymorphonuclear leukocytes. Abnormal white blood cells were not present. The bleeding time, coagulation time and platelet counts were normal. Blood calcium, the sugar tolerance curve and the determination of nonprotein nitrogen were normal. Blood cultures were negative. The blood Wassermann reaction was negative. Examinations of the stools revealed ova of *Ascaris lumbricoides* in only one specimen; otherwise, nothing abnormal was found. Skin and complement fixation tests for tuberculosis were negative. Cultures from the abscesses on the skin yielded *Staphylococcus aureus.* The rate of basal metabolism was determined on consecutive days and was — 20 per cent. Roentgen-ray examinations were without significance.

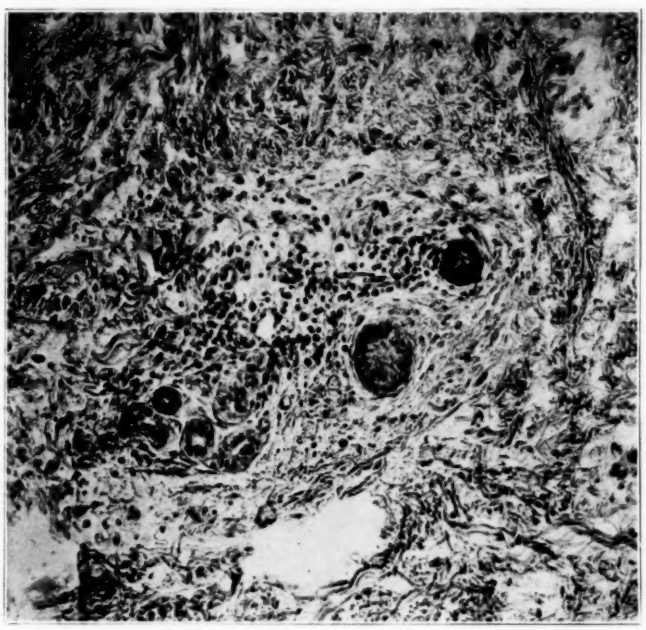

Fig. 3.—Corium of the skin, showing leukocytic infiltration and old granulation tissue in the region of the skin glands; low power magnification.

Biopsy was done, March 16, 1927. A section of skin, subcutaneous tissue and muscle was removed from the posterior aspect of the left thigh. Grossly, patho-

logic changes were not seen. Microscopically, the skin presented some blood pigment in the corium, which was a little more dense than normal. The lymphocytes and plasma cells were grouped about the sweat glands in part of the section (fig. 3). In the regions of this infiltration was a slight amount of proliferation of the connective tissue and small groups of bacteria resembling staphylococci (fig. 4).

Sections of the muscle showed localized collections of polymorphonuclear leukocytes, lymphocytes and larger mononuclears, some of which were plasma cells (fig. 5). Cocci similar to those in the skin were found here (fig. 6). In these regions there was a little proliferation of the connective tissue. Many of these lesions were near or surrounding small blood vessels. In some areas,

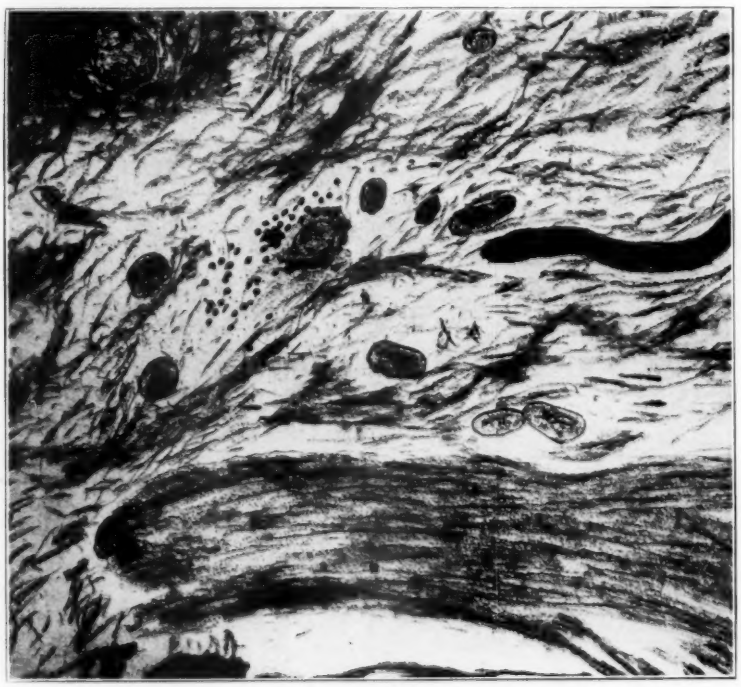

Fig. 4.—Camera lucida drawing of corium of the skin, showing cocci among the leukocytes and strands of connective tissue that vary in age; high power magnification.

the nuclei of the muscle cells were large and numerous and the sarcoplasm atrophic. In other groups of muscle cells the sarcoplasm was found in masses, taking the eosin stain and leaving the remainder of the sarcolemma filled with light-colored granular substance that did not resemble muscle (fig. 7). None of the nuclei were in the process of division. The diagnosis at biopsy was: chronic inflammation of the skin and chronic inflammation of the muscle, an infectious condition.

Course of Illness.—The patient was in the hospital ninety-four days, during which time practically no progress was made. The temperature ranged from

97 to 104 F. The highest elevations resulted from superficial abscesses which developed at intervals. These were incised, and the temperature returned to the usual elevation of 100 F. The average pulse rate was 110. Orthopedic measures were employed to overcome and prevent contractures. Ultraviolet and infra red rays were used with little advantage. Salicylates, calcium lactate, thyroid extract, cod liver oil and urinary antiseptics were administered at intervals, without definite results. Injections of sterile milk were used to stimulate general reactions. No change could be noted in the general or local condition until April 1, 1927, when a cellulitis of the neck developed followed by general sepsis and death three days later.

Necropsy (performed by Dr. A. J. Miller).—The body was poorly nourished and emaciated. About 50 per cent rigor mortis was present. Considerable lividity

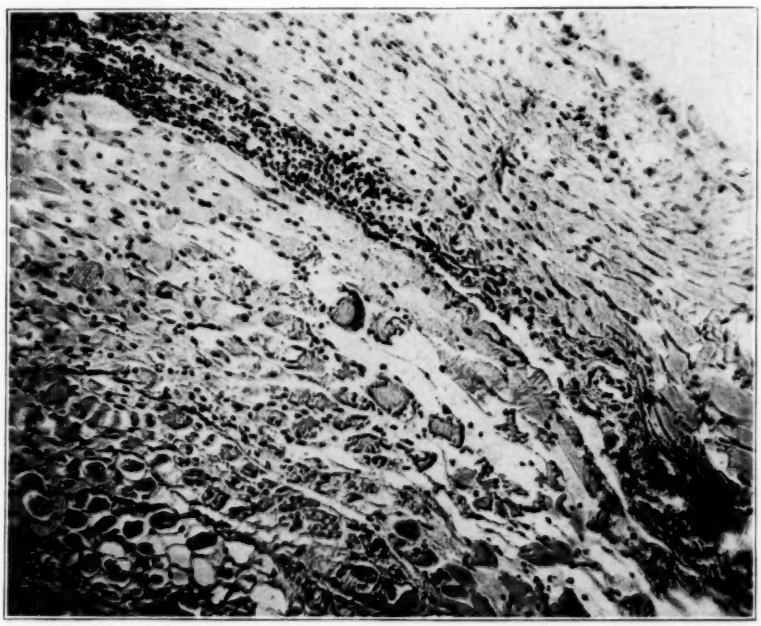

Fig. 5.—Biceps femoris muscle, showing dense infiltration of leukocytes in the septums, which tend to be grouped around the small vessels. The muscle in the upper part of the picture is atrophic; that in the lower portion is degenerated; low power magnification.

of the left ear, the left side of the face and the back was noted. There was an area of swelling in the region of the left parotid gland. The entire face was somewhat edematous. Around the knuckles of the hands, axillae and the elbows were numerous pitlike scars from 2 to 4 mm. in diameter. Over the chest and abdomen were numerous petechiae. Extension of both upper extremities at the elbow was limited to 135 degres. In the midline posteriorly over the sacrum there was a longitudinal unhealed wound 1.5 cm. long. On the posterior aspect of the left thigh was an unhealed wound 6 cm. long and 2 cm. wide, the site of the biopsy made three weeks before death. Slight edema of the extremities was present.

When the incision in the skin was made, little subcutaneous fat was encountered. The peritoneal cavity was exposed and the viscera found in normal relation. The peritoneal surfaces were smooth and glistening. It was estimated that there was 100 cc. of clear, straw-colored fluid in the peritoneal cavity. There was no excess fluid in the pleural or pericardial cavities. A small amount of thymic tissue was present in the upper mediastinum. The borders of the heart were normal. The thyroid gland was dissected out and was of normal size, color and consistency. The heart weighed 100 Gm. There were several petechiae in the pericardium, which was somewhat thickened. The heart itself was light red and firm. When a section was made, lesions were not noted in the valves. The aorta did not show any pathologic change.

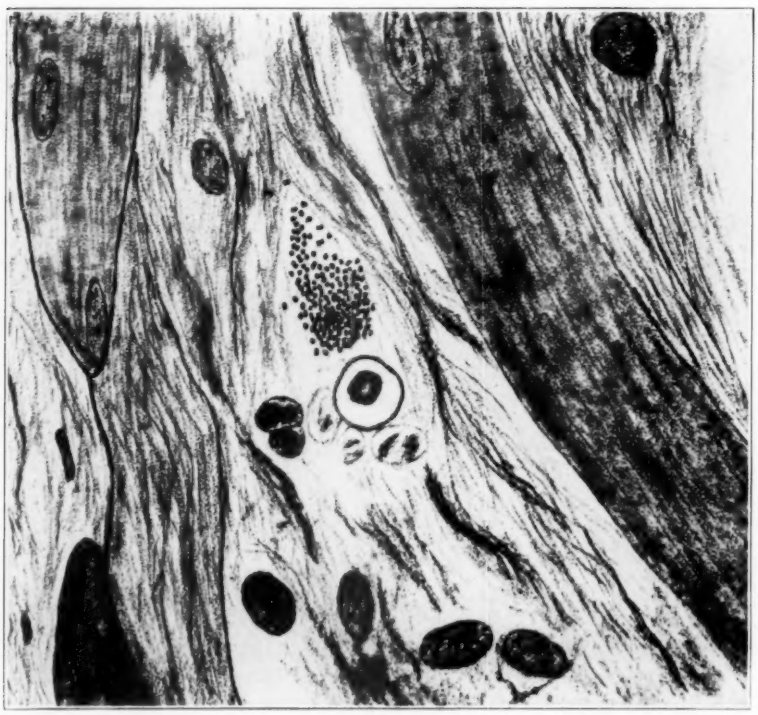

Fig. 6.—Camera lucida drawing of biceps femoris muscle, showing a large group of cocci close to a capillary containing an erythrocyte. Five leukocytes are in the field. The connective tissue varies in age. The muscle cells are swollen and have lost their transverse striae; high power magnification.

The left lung weighed 160 Gm. It was pale pink and was subcrepitant throughout. On sectioning, a thick, purulent material escaped from the bronchioles, but no areas of consolidation were found. The right lung weighed 190 Gm. and presented a gross appearance identical with the left.

The liver weighed 780 Gm. and was purplish. Over the right lobe were light-colored blotches. On sectioning, these blotches proved to be approximately 1 cm. in diameter and also were present within the substance of the liver. The liver was unusually firm. The lobules were indistinct.

The gastro-intestinal tract was opened from the cardiac end of the stomach to the rectum. Numerous petechiae were present in the mucosa of the stomach. On the gastric side of the pyloric valve was an ulcer 1.5 cm. in diameter, which extended through the mucosa into the submucosa. The intestines were thickened throughout the entire length. There was considerable hyperplasia of the lymphoid tissue. In the distal part of the ileum were several irregular ulcers. A papilloma 2 cm. in diameter was found in the cecum near the ileo-cecal valve. The mesenteric lymph nodes were enlarged throughout.

There was no evidence of pathologic alteration in the pancreas. The spleen weighed 110 Gm., was firm and cut with resistance. The sectioned surface was grayish-red, and pulp was abundant.

The right kidney weighed 75 Gm. The sectioned surface bulged slightly beyond the edge of the capsule. The capsule stripped readily, leaving a smooth

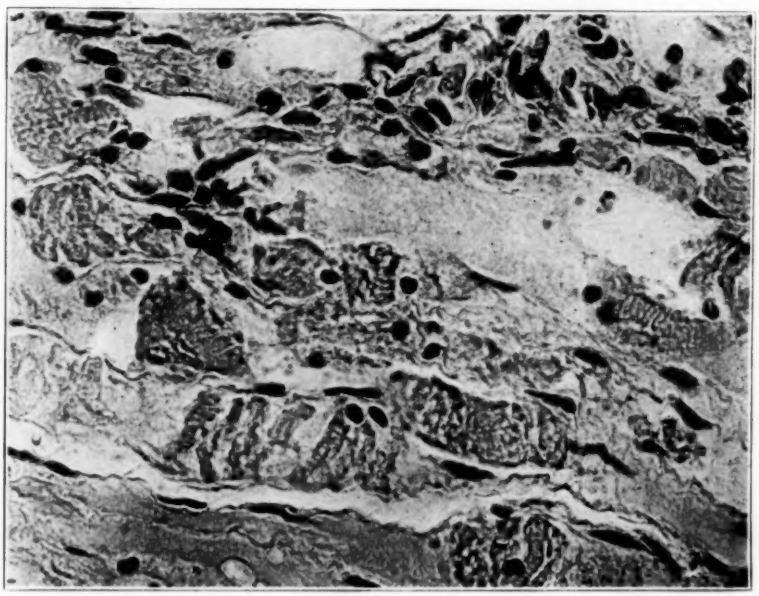

Fig. 7.—Biceps femoris muscle showing the sarcoplasm grouped into densely staining irregular masses leaving a portion of the sarcolemma filled with faintly staining, finely granular material. There are a few leukocytes between the muscle cells. Some of the connective tissue cells are recently formed; high power magnification.

injected surface. Purulent material was expressed from the pyramids. The cortical striae were prominent. The left kidney was similar to the right. A smear of the purulent material revealed no organisms, and most of the cells were kidney tissue cells. The ureters were found to be normal. The suprarenals were of normal size and consistency and did not show gross pathologic change. The bladder mucosa presented a few petechiae.

The prostate appeared normal. Gross pathologic change was not noted in the testicles. The left testicle was removed for microscopic study.

The skeletal muscle was extremely pale and appeared atrophic. The inguinal lymph glands were dissected out and found to be enlarged.

Microscopic Description.—The heart muscle showed some atrophy. In the left ventricle, the sarcoplasm was granular and stained faintly. The changes were similar to those caused by the toxin of infection.

Sections of the lung showed some edema and a small amount of hemorrhage. There were a few small localized areas of leukocytic infiltration, chiefly lymphocytes. The pancreas was normal. Sections of the liver showed congestion of the blood sinuses. Here and there in the septums were small collections of leukocytes. In some of these areas there was an increase in connective tissue, indicating damage to the tissues. These foci were suggestive of septicemia. The liver cells stained faintly, although few contained fat vacuoles. There was some blood pigment.

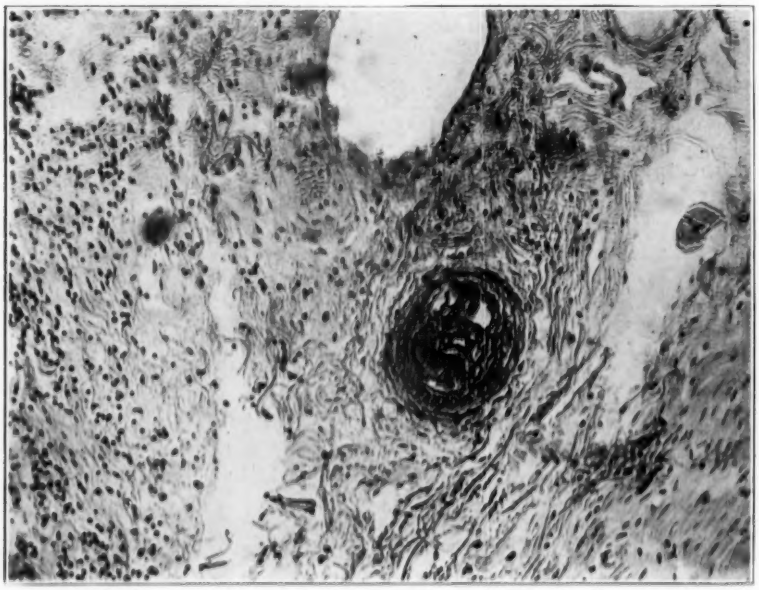

Fig. 8.—Wall of the stomach showing the thrombosed and canalized artery in the bed of the gastric ulcer. The left side, in which the leukocytic infiltration is most marked, is toward the mucosa. Probably the thrombosis and ulceration are in the relation of cause and effect; low power magnification.

Sections of the spleen showed considerable blood pigment. An increased number of polymorphonuclears were present in the sinuses. The sinus cells were sloughing and degenerating. Numerous mitotic figures were seen in large pulp cells.

One lymph node from the mesentery was examined. The blood vessels were congested. The lymph follicles were small and showed much hyperplasia of the stroma. The germinal cells showed considerable degeneration. In the sinuses were many large mononuclears showing degeneration. Blood pigment could be seen in the peripheral sinuses. In the hilum several large masses of bacteria were seen, some of which were in the lymph spaces and follicles.

The kidneys showed only a few sclerosed glomeruli. Most of the parenchymal cells were granular and swollen, and in places desquamation was present. The pyramidal portion presented localized congestion and some desquamation. No abscesses were found. The changes in the kidneys were less than would be expected after purulent exudate had been found in the pelvis.

Sections of the stomach were taken through the ulcer at the polyrus. The necrosis involved part of the muscle of the sphincter. Some undermining of the stomach side of the ulcer was noted, indicating that the lesion was an old one. In the floor, at about the center of the ulcer, was a thrombosed artery. As there was considerable organization and one small canal, the thrombus may be looked on as an old one and a probable cause of the ulcer (fig. 8).

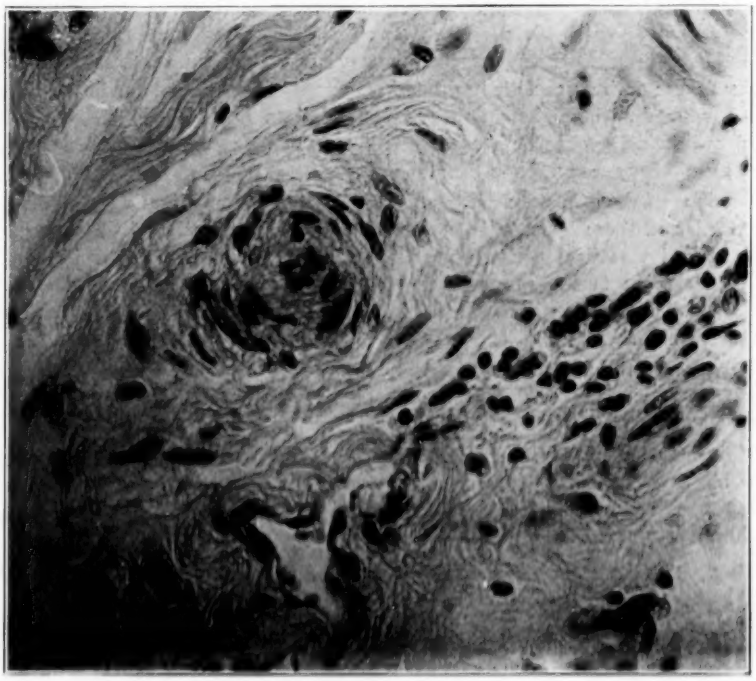

Fig. 9.—Skeletal muscle septum. One small blood vessel is entirely occluded by scar tissue. Plasma cells and lymphocytes have collected in the vicinity; low power magnification.

A section of the small intestine was taken through the lesion mentioned in the gross description. The lymphoid tissue was swollen, congested and densely infiltrated, and the germinal centers were breaking down. The mucosa was ulcerated down to the musculature. In the submucosa at the site of the lesion was a thrombosed arteriole. Near it was another vessel that was thrombosed and organized with canalization. Probably these occlusions were the cause of the lesions in the mucosa.

The cells of the suprarenals stained faintly, and considerable vacuolization was present.

Sections of the prostate showed some cellular infiltration in the region of the prostatic glands and also in the mucosa of the bladder.

The thyroid tissue was normal. Sections of the testicle were not remarkable.

Sections of muscle were taken from the pectoralis major, biceps femoris and the rectus abdominalis. They all showed simple atrophy, and in addition some of the sections revealed marked degeneration. This consisted of heaping of the sacroplasms in large, densely staining, hyaline-like masses, which left the rest of the cell pale or clear. In the region of these changes were some leukocytic infiltration and an increase in connective tissue. Here and there throughout the sections were localized areas of leukocytic infiltration consisting of plasma cells, lymphocytes and large mononuclear phagocytes with only an occasional polymorphonuclear leukocyte. In these regions was considerable granulation tissue which varied in age. Most of these regions were in the immediate vicinity of small blood vessels. Many of the blood vessels were thick-walled, and some of them were entirely occluded (fig. 9). One vessel was found canalized. These lesions seemed to be definitely those resulting from infected emboli. By means of special stains, groups of cocci were found. These resembled staphylococci in their morphology and arrangement.

Sections of the skin were examined. The fibrils of the corium were moderately separated, and there appeared to be some new connective tissue. In the regions of the blood vessels and sweat glands, small masses of leukocytic infiltration similar to those in the muscle were frequently seen. There were also great numbers of cocci. Small masses of old blood pigment were frequently found in the corium.

Sections of the material obtained at biopsy were reviewed with special stains, and identical changes in the tissue with similar cocci were found in the tissues removed three weeks before death.

Cultures of the blood, spleen and muscle were made at the time of necropsy. The blood contained *Staphylococcus aureus* and *Streptococcus hemolyticus*. Cultures of the spleen contained *Streptococcus hemolyticus* and *B. coli*. Cultures of the muscle remained sterile.

Anatomic Diagnosis.—The diagnosis was: emaciation; scars and pigmentation of the skin; an unhealed surgical wound 3 weeks old; muscle contractures of the extremities; petechiae in the skin, pericardium, mucosa of the stomach and bladder; embolic ulcers of the stomach and ileum; chronic inflammation of the liver and spleen; acute splenitis; focal lesions of infection in the skin, muscle, liver, lung and lymph nodes; cloudy swelling of the kidney; generalized atrophy of skeletal muscle; ascites and edema, and septicemia.

COMMENT

The patient's condition was primarily one of infection. Evidence of this is the fact that organisms were found in the material obtained at biopsy and also that the lesions in the tissues resembled those produced by infectious agents. The offending organism was undoubtedly the staphylococcus. The streptococcus was considered a terminal invader. This localization in the skin is common, but the localization in the muscle is not common. The primary lesion was not demonstrated.

SUMMARY

1. Clinically, there was a gradually developing septicemia with especially marked involvement of the skin and muscles. This was manifest in the skin by infiltration, formation of abscesses and pigmentary change and in the muscles by atrophy and resulting contractures of the joints.

2. Biopsy and autopsy showed general septicemic changes.

3. The finding of staphylococci in the muscles and skin in material obtained at biopsy is suggestive of the etiology, though experimental work was not done to prove the specificity of the organisms.

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MYATONIA CONGENITA OF OPPENHEIM

REPORT OF A UNIQUE CASE *

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Myatonia congenita of Oppenheim, also called amyotonia congenita, is not now considered as rare as it was formerly thought to be, because of its more widespread recognition. Reuben [1] reviewed the literature in 1917 and found reports of 136 cases. When Haushalter [2] reviewed it again in 1920, he found reports of 155 cases.

A point that has been mentioned in connection with this particular disease by Holt and Howland,[3] must be kept in mind; namely, that when a disease is first discovered, only the most striking and complete cases are recognized; later, incomplete syndromes are identified. It is not my intention to review the literature or to discuss in detail all phases of the disease, as this has been ably done, but to present a case of a partial syndrome with a unique contracture, and to show the profound consequences that result from this absence or nonfunction of the anterior flexor muscles of the neck.

REPORT OF CASE

History.—E. I., a boy, aged 7, was brought to the clinic because of breathing through the mouth and difficulty in walking.

The family history was interesting. The paternal grandfather died at the age of 64 of paralysis of the throat; the paternal grandmother died at 56 of "brain fever," and the maternal grandfather at 50 of blood poisoning. The maternal grandmother and the parents were well. The first child of the parents was stillborn at seven months, the "spine being open and the top of the head missing." The second pregnancy ended in abortion at three months, and the patient was the product of the third conception. He had a brother 4 and one 3 years of age. The sixth pregnancy resulted in a stillbirth at eight months, the physician in attendance stating that the infant was "like the patient but worse, with a very small neck." There was no history of chronic illnesses or of consanguinity in the family.

The patient had had measles and an operation for adenoids, but no other illnesses and no accidents. The birth was normal; instruments were not used. He was breast-fed for seven months, acquired his teeth normally, but was never able to hold his head up as he should. He took a few steps at 1 year and at 14

* Received for publication, Sept. 16, 1927.

* From the Neurological Department of the Battle Creek Sanitarium Clinic.

1. Reuben, M. S.: Amyotonia Congenita of Oppenheim, Arch. Int. Med. **20**:641 (Nov.) 1917.

2. Haushalter, P.: Amyotonia Congenita: Oppenheim's Disease, Arch. d. méd. d. enf. **23**:133 (March) 1920.

3. Holt, L. E., and Howland, John: Diseases of Infancy and Childhood, ed. 7, New York, D. Appleton & Co., 1920, p. 827.

months walked with a peculiar unsteady gait. He learned to talk normally, but growth in stature had been slow since the age of 5.

When seen, the boy was attending school regularly, was always near the head of his class, enjoyed intellectual work, but played poorly at physical games. He walked with a ducklike waddle, bent forward at the hips, but held his head back. The normal swing of the arm was voluntarily suppressed, while the shoulders attempted to execute the automatic associated movements. During walking, the arms were not allowed to hang vertically but were thrust backward in line with the trunk. In walking down the stairs, he invariably placed both feet on each step, fearing to fall forward and always accepting help from others to maintain his balance. There was no peculiarity in rising from a reclining position. He had a stupid expression and an open mouth. Before I knew his history, my first impression was that he was an idiot.

Fig. 1.—Moving picture to show characteristic gait. The large head of peculiar shape, the open mouth, small neck, poor musculature, stooped posture and the absence of normal arm swing which is voluntarily suppressed should be noted.

Examination.—On examination he proved to be 46½ inches (118 cm.) tall, with the pubis in the middle of the body, and weighed 40 pounds (18.1 Kg.). The osseous system was of good proportion though it was small throughout, except the head, which was relatively large. Maxillary prognathism was present. The middle toe of each foot was slightly longer than the second. Considerable lordosis was present, and the child had abnormal mobility of all joints of the body except the knees and the neck in which contracture was present.

All the muscles were small, especially those of the neck the posterior group of which was in a state of contracture from unopposed action. The neck was 9¼ inches (23 cm.) in circumference. There was no evidence of the sternocleidomastoid muscles; cephalic rotation was extremely weak and difficult. When

resting on his back, the patient was unable to rotate his head at all but moved his eyes to look to the sides. He could not bend his head forward except with the aid of his hands.

The skin was warm and moist except on the feet, where it was cold. Axillary and pubic hair was absent. The thyroid was not palpable, but glands were felt in the right inguinal region. The tongue was badly coated; the tonsils were large and infected, though not with virulent bacteria. Examination of the chest showed dulness on percussion and râles in the lower left side. The temperature was 100 F. (Evidently, an acute condition of the lungs was present.) A roentgenogram confirmed this observation. There was no evidence of persistent thymus. The pulse rate was 120, but the heart was normal. Abdominal abnormality was not found, but the boy had definite phimosis.

Figure 2

Figure 3

Fig. 2.—Best effort of patient to look to right. Absence of rotation of the head should be noted.

Fig. 3.—Absence of sternocleidomastoid muscle which should be tense on the proximal side.

Examination of the teeth showed marked caries. The Kolmer test with the blood was negative. The nonprotein nitrogen amounted to only 21.4 mg. per hundred cubic centimeters of blood; aside from this, results of the blood chemistry, the blood count and both the gross and the microscopic examinations of the urine were normal. A stereogram of the head and neck was described as follows: "The bones of the calvarium are thinner than usual and the channels for some of the cerebral vessels are unusually prominent. This is especially pronounced in the frontal region. There are also some digital markings on the inner table particularly in the occipital region. This condition would suggest increased intracranial pressure. The sella turcica is unusually small, at least a third smaller than would be

expected in one of the patient's age. The sinuses are well aerated and show no roentgen-ray evidence of any inflammatory process. The cervical spine is normal." Examination of the fundus of the eyes revealed nothing abnormal.

All deep reflexes were absent, but on reinforcement they were obtained. This diminution seemed to be due to marked flaccidity and relaxation. Electrical tests were qualitatively normal. Strong currents (necessary for a contraction) were well tolerated. The myasthenic reaction of Jolly was absent.

COMMENT

There is a growing recognition of two forms of myatonia congenita. The more common form occurs in one member of a family and is malignant in that the patients usually die in early infancy; the other form,

Figure 4

Figure 5

Fig. 4.—"Normal" posture when standing. This figure should be compared with figure 5.

Fig. 5.—The strength of the spinal muscles is sufficient but the head must be held forward to avoid the overbalancing effect of its dropping backward.

about one-tenth as common, is familial and relatively benign. The patients of the latter group usually survive to the age of puberty and may even recover. It is to the latter group that the case here presented belongs. It seems that though the parents of this patient apparently were entirely well, the products of their union were often faulty. If this case adds anything to the understanding of myatonia congenita, it seems to add strength to the idea that the pathologic condition is a faulty or incomplete development.

No definite cause of the intracranial pathologic alterations was found, but the mechanism of the gait and the production of the stupid facies are interesting. These were based on the absence, or at least the practical absence, of the anterior muscles of the neck. The first result of this absence was the retraction of the nuchal muscles. As the patient attempted to stand erect, his head dropped backward; he momentarily lost coordination and fell backward. To counteract this, he would lean forward, bringing the center of gravity of the head anterior to the pivotal point so that the force of gravity helped hold it forward rather than backward. When he clasped his hands behind his head to hold it forward, he could stand easily (figs. 4 and 5). Because the head was constantly held back, the muscles of the floor of the mouth were tightened and kept the mouth open. The lower jaw thus did not develop, and maxillary prognathism resulted. The absence of the sternocleidomastoid muscles made cephalic rotation difficult and the apparent rigidity of the neck and the consequent movements of the eyes gave the patient a bizarre appearance. The arms were thrust backward when walking, apparently to avoid excessive swing, but were allowed to hang vertically during repose.

The incapacity was crippling and rendered the boy a freak. Transplantation of muscle has been considered but will be delayed in the hope that improvement will give rise to muscles of sufficient size. Perhaps part of the pectorales majores may be utilized for this purpose.

Without an operation or autopsy in this case, the actual absence of the flexor muscles of the neck and the sternomastoid muscles cannot be predicated, but the movements that those muscles should execute are impossible.

The characteristics on which the diagnosis was made and which eliminate the question of a mere regional aplasia are: (1) the generalized small musculature, (2) marked muscular flaccidity, (3) absence of all deep reflexes except on reinforcement, (4) normal qualitative electrical reactions, with (5) unusual tolerance for electrical stimulation, (6) absence of pseudohypertrophy, irregular involvement and progression of the disease, (7) gradual improvement, (8) blank facies, (9) presence of contracture and (10) abnormal mobility of all joints except those involved in contracture.

Talipes equinus and contracture of the limbs of various kinds are often seen in myatonia congenita, but no record of contracture of the neck, such as occurred in this case has been found.

AMAUROTIC FAMILY IDIOCY (TAY-SACHS TYPE)

A CASE WITH A PROTRACTED COURSE *

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As rich as the literature dealing with various types of amaurotic family idiocy undoubtedly is, new cases occasionally occur which are of such unusual and great interest that their recording is fully justified. The present case, for instance, possesses the following remarkable features: (1) the onset was late and the duration unusually long, three years and nine months; (2) the retina lacked the cherry-red spot, in the presence of all the other cardinal features of the infantile type of amaurotic family idiocy; (3) pathologically, the condition proved to be a transition from the infantile to the late infantile form and (4) it showed, in addition to typical microscopic changes, a number of macroscopic changes that might be considered equally typical.

REPORT OF CASE

Clinical Report of Case (Dr. Parmelee).—D. L. entered the Presbyterian Hospital, April 21, 1926. He was born March 7, 1923, the first child of apparently healthy young parents. The father and mother were Polish Jews and first cousins. The mother's pregnancy and labor were normal, the latter lasting only two hours. The infant was not asphyxiated or cyanotic; he did not have convulsions during the first two weeks of life; he seemed normal in every way. He was fed at the breast until he was 3 months old, at which time he weighed 9 pounds (4,082 Gm.); the weight at birth was 6½ pounds (2,954 Gm.).

When he was 3 months old, a lump was noticed on his back; this gradually became more prominent. Roentgen-ray examination made when he was 10 or 11 months old suggested Pott's disease, and he was put in a plaster cast for three months. He was then in braces for a while, and later was put in a cast again but remained in it only a short time.

At 13 months of age, he was able to stand alone and made some attempts to walk, but even at 2 years of age was only able to take a few steps alone. When he was 2½, an orthopedic surgeon diagnosed the condition as tuberculous spondylitis and again put him in a cast, in which he remained for ten months.

It was during this period that the parents began to notice retrograde mental changes. They had never felt that he was as bright as other children

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* From the Pediatric Department of the Presbyterian Hospital, the Pathologic Laboratories of the Research and Educational Hospital and the Illinois State Psychopathic Institute.

of his age that they knew, but he had seemed alert and happy, took an interest in his toys, seemed to comprehend things that were said to him and could say "mama" and "papa." He took less and less interest in his surroundings and also did not seem to see objects held in front of him. He gradually grew worse, and when I first saw him in April, 1926, he seemed to be practically blind; he could not focus the vision on anything and did not seem to comprehend anything that was said to him. However, he was particularly sensitive to sound, and the parents said that for the last six months he would waken with a start at any unusual noise, as when something was dropped to the floor, even though he appeared to be almost constantly drowsy.

Such a condition has not occurred on either the father's or the mother's side of the family in theirs or a previous generation, but they now have a second child, a boy, aged 2 years 3 months, who is apparently going through the self same course as the brother here recorded. A lump was noticed on his back by the mother when he was 3 hours old; he has never been able to take more than a few steps alone and, while he is definitely brighter than his brother, he is not as alert as he should be for his age and is falling behind rather than advancing in mental development.

Physical Examination.—The child had a large frame, was fairly well nourished, seemed helpless and did not appear to see or take any interest in his surroundings. He did not stand or sit alone but moved all his extremities. Whenever handled or spoken to, he cried and became tense and spastic. His skin was dry and scaly, especially over the trunk, and resembled ichthyosis. His breathing was stertorous and noisy, more markedly so whenever he was disturbed, and his mouth was constantly open.

Marked nystagmus was present; the pupils did not react to light nor in accommodation; transient strabismus was noted. There was some mucopurulent discharge from the nostrils; the teeth were small and widely spaced; the pharynx was somewhat inflamed.

The lungs were resonant throughout; areas of dulness or râles were not present, but harsh noisy inspiratory sounds from the throat could be heard. The heart was normal in size, rate and rhythm, and no murmurs were heard. The abdomen was rounded and somewhat tympanitic, but was not rigid. The liver was palpable three fingerbreadths below the costal margin. The spleen was not palpable. The genitalia were normal except that the left testicle was not in the scrotum.

Neurologic Examination.—All the deep tendon reflexes were greatly exaggerated. The Babinski sign was present on both sides, also a definite sustained ankle clonus. The Chvostek sign was present. The neck was slightly rigid. The superficial abdominal reflexes were faintly positive but delayed. Sensation to pain was readily elicited by pin pricks. A marked kyphosis was present in the region of the second and third lumbar vertebrae, with a lumpy protruberance to the left.

The eyes were examined by Dr. Allen, an ophthalmologist, on April 22, and his report follows:

"Baby lies on his back rolling the head and eyes about in an aimless fashion, occasionally fixing the vision. Media clear. In the posterior part of each eye there is a yellowish nebulous appearance of the retina. On account of the nystagmus, nothing more definite could be determined. A cherry-red spot was not seen, but should this have been present six to twelve months ago, it would not now be found. From an eye standpoint this is very suspicious of amaurotic family idiocy."

Roentgen-Ray Examination (Dr. Rose).—On April 22, a roentgenogram of the spine showed little scoliosis of the lumbar region with a distinct narrowing of the body of the second lumbar vertebra. On lateral view, this body was considerably smaller than the others and was somewhat wedge-shaped. There was a distinct kyphosis of the spine at this region.

A roentgenogram of the skull showed a spherical outline; however, this was not definitely abnormal. The sella was within normal limits.

Examinations of the Blood and Spinal Fluid.—The spinal fluid was not under increased pressure. Examination showed: sugar, 80.6; calcium, 7.22; cell count, 7; Nonne-Apelt reaction, positive. The Wassermann reaction was negative. The colloidal gold test was 0012210000.

The blood count showed: hemoglobin, 90 per cent; red blood cells, 5,400,000; index, 9; white blood cells, 17,000; the differential count showed: neutrophils, 59; small mononuclears, 6; large mononuclears, 30; transitionals, 3 and eosinophils, 2. The Pirquet test was negative. The temperature varied between 99 and 101.

The patient was in the hospital from April 21 to April 23.

Subsequent Course.—I saw the child about every month with substantially the same symptoms, except that the spasticity became more marked and the stupor more intense.

On Dec. 4, 1926, he became acutely ill following an infection of the nose and throat and was again admitted to the Presbyterian Hospital. The temperature rose rapidly to 104 and finally reached 106.8. Respiration became more and more difficult and the color more and more cyanotic. He remained in a constant state of spasticity and opisthotonos, and finally died on December 6. Autopsy was done on the same day.

Observations at Necropsy (Dr. C. W. Apfelbach).—The condition was diagnosed as marked atrophy of the cerebral cortex and atrophy of the flexor muscle of the legs. The body was that of a white child, weighing 27½ pounds (12.4 Kg.). The body nourishment was good. Body heat was still present, and rigor mortis was beginning. The trunk was large in comparison with the extremities. The finger tips, when the arms were extended, reached to the level of the perineum. The anterior-posterior diameter of the chest equalled the transverse diameter. The abdomen bulged at the sides. The legs were thin in comparison with the trunk and arms. The feet were markedly extended. A deformity of the spine was represented by scoliosis of the lower thoracic and all of the lumbar portion, the bend extending about 3 or 4 cm. to the left of the midline and the spine bulging toward the left and backward. The skin of the front of the legs and top of the feet was covered by large, light brown superficial scales. The skin of the trunk was also desquamated in many places. The sclerae were light gray, almost white. The hair of the scalp was abundant, brown and curled.

The dura was adherent along the superior longitudinal sinus and stained pale red. The pacchionian bodies were numerous along the superior longitudinal sinus. The convolutions at the top of the brain were narrow, the sulci widened so that they were from 4 to 5 mm. wide and formed a continuous network of blue in the surface of the brain. In the back half, the leptomeninges were raised above the level of the convolutions by clear fluid. The cerebrospinal fluid was clear. The cerebellum was symmetrical; it measured about 2 cm. across and 1 cm. from top to bottom, exposing a large fourth ventricle. The

convolutions of the occipital lobe were small; most of them were about 8 or 10 mm. wide, and in their surface there was a superficial groove from 1 to 2 or 3 mm. deep. The occipital lobes were small, roughly about 3 or 4 cm. long, and protruded as small appendages from the parietal lobes.

The greatest amount of atrophy measured by the size of the convolutions in order was in the occipital lobe, the back portion of the parietal lobe and then the frontal lobe, the temporal lobe having the largest convolutions. The brain weighed 745 Gm. An abundance of spinal fluid dripped from the brain while it was being examined.

The calvarium was 16 cm. long and 13.5 cm. wide. About one third of the calvarium transmitted light. The upper margins of the petrous portions of the

Fig. 1.—The cerebral hemispheres. The occipital lobe is atrophied, leaving a portion of the cerebellum uncovered. Through the thickened pia, the dilated sulci and the atrophied convolutions are clearly seen. *Inp. f.* is the interparietal fissure.

temporal bones were rounded and superficially grooved. The posterior fossae were large, extending down and forward close to the groove for the lateral sinus in front. In the petrous portions of the temporal bones, an abundance of mucus was found. In the cells were gray soft places resembling granulation tissue.

The ear drums on both sides were intact, smooth and shiny. There was no change in the intraventricular disk.

Macroscopic and Microscopic Examination (Dr. Hassin).—The pia was opaque throughout and was greatly thickened, but not adherent to the brain. The

surface of the brain was irregular and grooved, because of the dilated sulci that separated the markedly atrophied convolutions. As noted, the occipital lobe was especially involved — it was reduced to one-third its size, leaving a part of the cerebellum uncovered (fig. 1). The inferior portion of the cerebellum was greatly excavated and depressed, with especial involvement of the inferior vermis, a portion of the tonsils and the biventral lobes. The depressed or excavated areas were bridged by a thickened arachnoid, as shown in figure 2. The lamellae of the cerebellum were thinned, widely separated from one another and were firm to the touch and on cutting. Though the atrophied condition of the convolutions and the depth of the gaping sulci were evident with the pia unstripped, they were much more so with this membrane removed. Figure 3, for instance, shows the convolutions of the frontal lobe

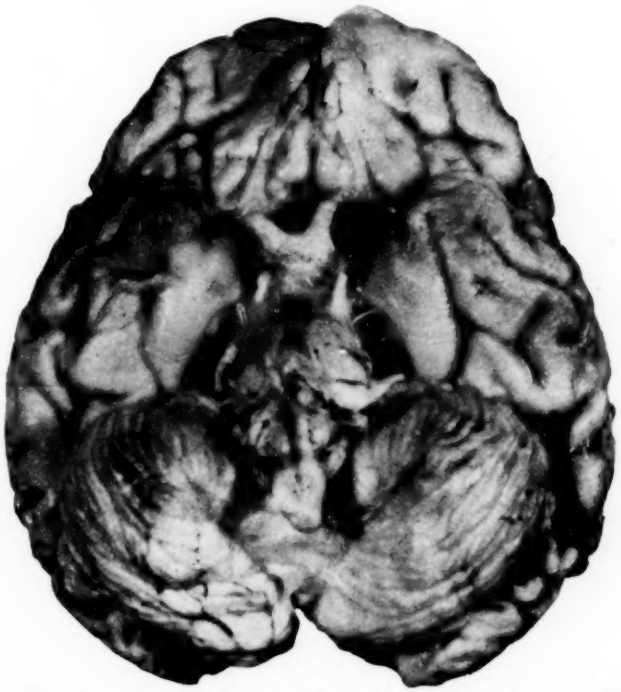

Fig. 2.—The base of the brain. The middle part of the cerebellum is excavated and bridged by a hyperplastic arachnoid membrane. The lamellae of the cerebellum are thinned; the convolutions of the temporal lobe appear rather wide.

to be atrophied and the sylvian fissure gaping with a portion of the atrophied island of Reil exposed. In figure 1, a like atrophy of the occipital lobe may be seen through the pia-arachnoid; the interparietal fissure was gaping, while the temporal convolutions, in contrast, were wide (fig. 2) and only some sulci were prominent. The spaces between the atrophied convolutions were filled with a hyperplastic pia and were bridged by a thickened arachnoid, while the ventricles of the brain, including the third, were greatly dilated (fig. 4). The optic thalami and the corpus callosum were considerably atrophied, and the whole brain was, like the cerebellum, hard to the touch.

Fig. 3.—Frontal lobe separated from the temporal lobe by a gaping sylvian fissure (*S. f.*). The atrophied convolutions and deep sulci are clearly seen. The longitudinal fissure also is gaping.

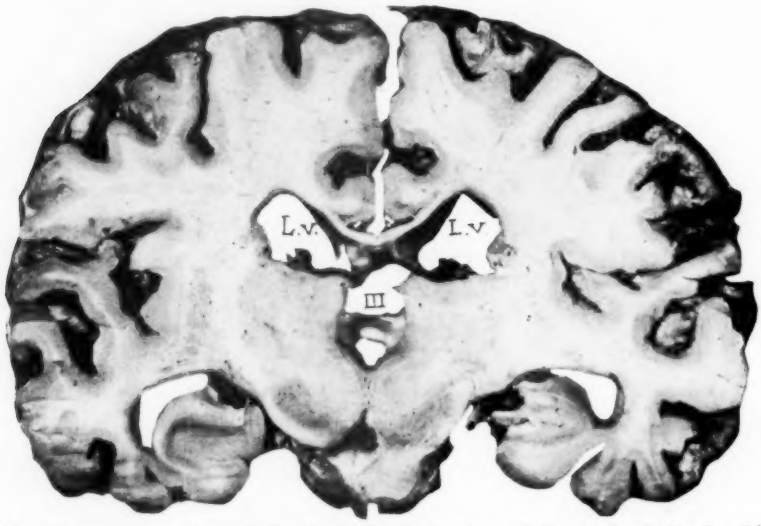

Fig. 4.—Cross-section of the brain in the region of the optic thalami, which are considerably atrophied; the third venticle (*III*) is dilated like the lateral ventricles (*L. v.*); the sulci are deep, wide and filled with a hyperplastic pia and bridged by a thickened arachnoid; the corpus callosum is also greatly atrophied.

The gross changes may be defined as diffuse atrophy of the brain, with especial involvement of the occipital lobe, cerebellum and the optic thalami.

Studies of various portions of the brain, in celloidin, paraffin and frozen sections and stained with numerous methods, revealed universal changes in the cells, types of which are shown in figures 5 and 6. Figure 5 represents a Purkinje cell with swollen dendrons and a number of distended formations in the molecular layer (marked *x*). These expansions are also abnormal, swollen dendrons of Purkinje cells which were filled with a substance that stained dense blue with the hematoxylin (Weigert-Kulschitzky) method and gave a pale orange stain with scarlet red. Other staining methods, such as toluidin blue, always revealed swelling of the ganglion cells, expansion of

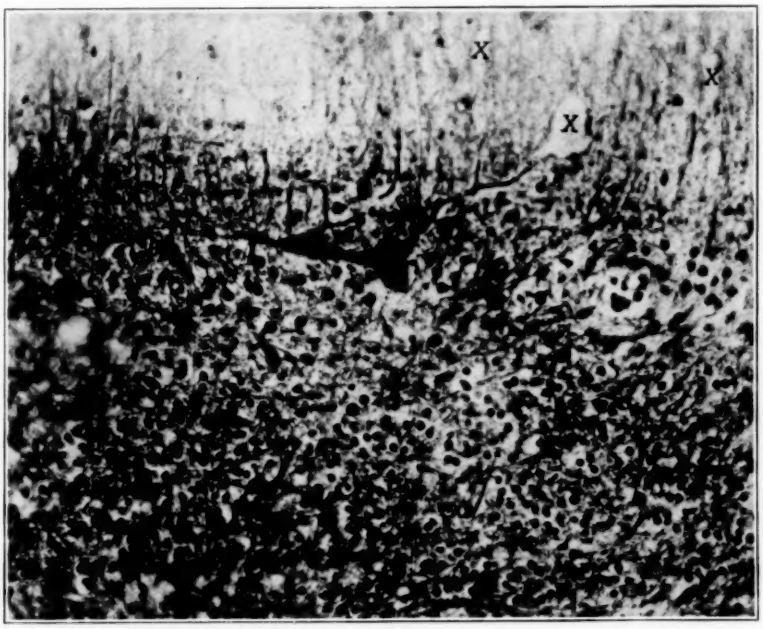

Fig. 5.—Lateral cerebellar lobe. In the center is a Purkinje cell with visible dendrons; the left is considerably expanded; swollen dendrons (not in focus) are also seen in the molecular layer (*x*). The lower part of the illustration represents the granular layer of the cerebellum which appears rarefied and in which, especially to the right, a number of nerve fibers are seen (so-called mossy fibers); the basket and climbing fibers accompanying the dendrons cannot be seen. Bielschowsky stain; × 275.

their processes, absence of Nissl bodies and of such phenomena as satellitosis and neuronophagia. The afferent cerebellar nerve fibers (mossy, climbing and basket) were preserved (fig. 5); the granular layer was slightly rarefied, resembling in some sections the one seen in the late infantile type of amaurotic family idiocy. The glia was proliferated and was abundantly represented by the so-called cytoplasmic and fibrous variety. Figure 6 represents the types of the ganglion cells in the rest of the brain. The cells were almost always balloon or pear-shaped, the majority of the dendrons reduced in size or

swollen, the neurofibrils and the nucleus misplaced to the periphery and the Nissl bodies lacking and replaced by a dust-like amorphous substance enclosed within the endocellular network. Such a far advanced destruction of the cells was especially in evidence in the occipital lobe and the optic thalami, while in the pons and the region of the peduncles, including the nucleus ruber, the changes in the cells were less marked. They stained only slightly with Weigert-Kulschitzky's method (fig. 7), and did not show lipoid substances; only the occipital lobe and the optic thalami showed cells with a great mass of granules that stained bright red with the scarlet red staining method and brownish with osmic acid. In these two portions of the brain the changes

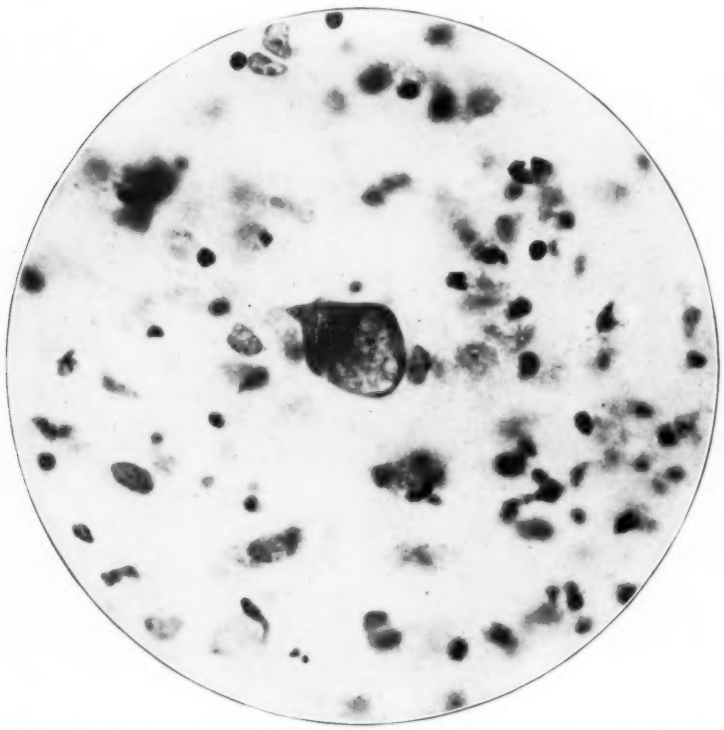

Fig. 6.—In the center of this illustration is a ganglion cell, showing an endocellular network in the swollen body, a nucleus misplaced to the apical dendron. Many other ganglion cells are not in focus and show only their nuclei; the dark scattered nuclei are what is known as oligodendroglia. Toluidin blue; × 600.

in the glia were, as in the cerebellum, of a proliferative nature and resembled in some instances a so-called glia scar tissue. In contrast to such wide-spread changes in the cells, the nerve fibers, in proportion, were much less involved. Many nerve fibers were well preserved and stained well with the method of Weigert-Kulschitzky (fig. 7) and similar methods of staining with hematoxylin, but as a rule the density of the nerve fibers in the brain was much reduced; that is to say, the nerve fibers were sparse, often thinned and stained pale. In other regions, such as the medulla oblongata, peduncles, pons and corpus

striatum, the nerve fibers were well represented and well stained; in the corpus callosum and in the strata of the cortex (frontal, occipital) occupied by the tangential fibers, they were absent altogether (fig. 7).

In the corpus callosum, for instance, the nerve fibers (fig. 8) were replaced by immense numbers of so-called gitter cells, few of which contained lipoids. The majority contained prelipoids and, as figure 9 shows, the perivascular spaces were packed with large amounts of lipoid substances (neutral fats) into which the prelipoid contents of the scattered gitter cells are supposed to transform while being transported to the blood vessels.

Among the white nerve fibers in some regions of the brain (occipital, temporal, pons and lenticular nucleus), a number of cell bodies, much resembling gitter cells, were scattered singly or in groups. They were always

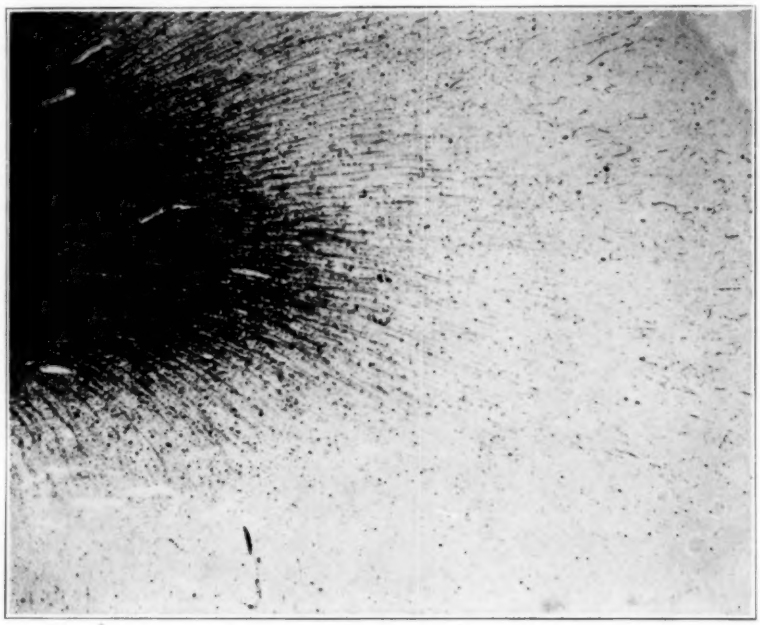

Fig. 7.—Absence of the intracortical tangential fibers in the cortex; poverty of myelinated fibers in the subcortex and their abundance in the corona radiata. The ganglion cells in the cortex may be seen with a hand lens as pale blue bodies containing few so-called hematoxylinophilic granules; the small dark round bodies are gitter structures filled with catabolic products. Frozen sections stained with Weigert-Kulschitzky.

packed with a granular substance and stained densely blue with Weigert-Kulschitzky's hematoxylin (fig. 7); they stained metachromatically reddish with toluidin blue, and did not stain with scarlet red or silver nitrate stains. The substance as noted was granular, filling the small vacuoles of the cell body and pushing the nucleus that was rich in chromatin to the periphery. In many instances the metachromatic substance was found also over the cytoplasm of the astrocytes and in especially large amounts near the blood vessels or within their adventitial spaces (fig. 10). None were found in the sub-

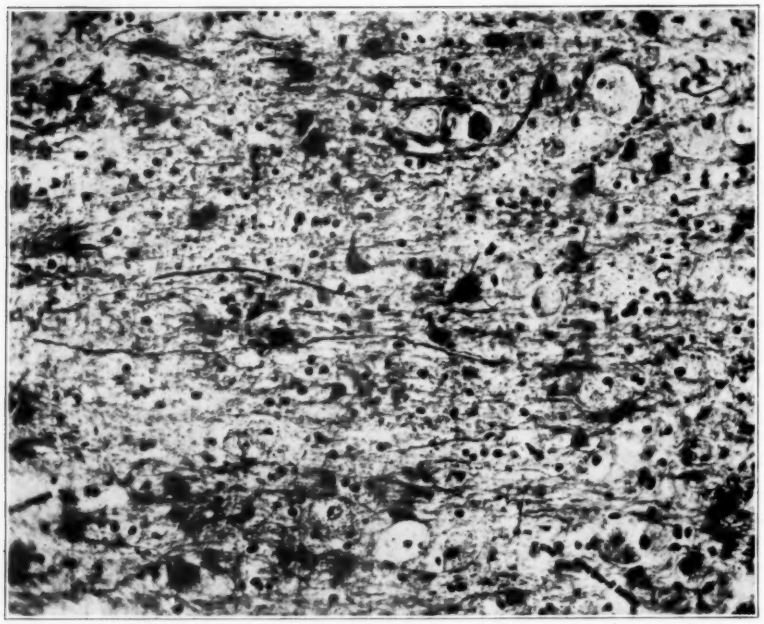

Fig. 8.—Corpus callosum. Few nerve fibers are present; they are shown here in the form of naked axons (black lines) the dark bodies supplied with processes are cytoplasmic glia cells; the larger round pale formations, somewhat granular in appearance, are gitter cells. Bielschowsky stain; × 275.

Fig. 9.—Corpus callosum. The black masses are lipoids within perivascular spaces of blood vessels and capillaries. Scarlet red; × 60.

arachnoid space. The foregoing substances, described by Alzheimer as basophil-metachromatic, are not lipoids but are prelipoids or rather the preliminary stages of the lipoid or prelipoid degeneration of the central nervous system. Alzheimer[1] looked on them as catabolic products (Abbaustöffe) capable of being transformed into fat and transported to the blood vessels for final elimination. These substances are evidently identical with what the French authors and in this country Kraus[2] and Bailey described as mucinoid or

The blood vessels exhibited normal walls, prominent endothelium and adventitial cells. Their perivascular spaces were frequently dilated and harbored various elements, mostly gitter cells packed with lipoids, prelipoids and other substances. In some instances the cells in the perivascular spaces were so dense (fig. 10) that the condition greatly resembled an inflammation.

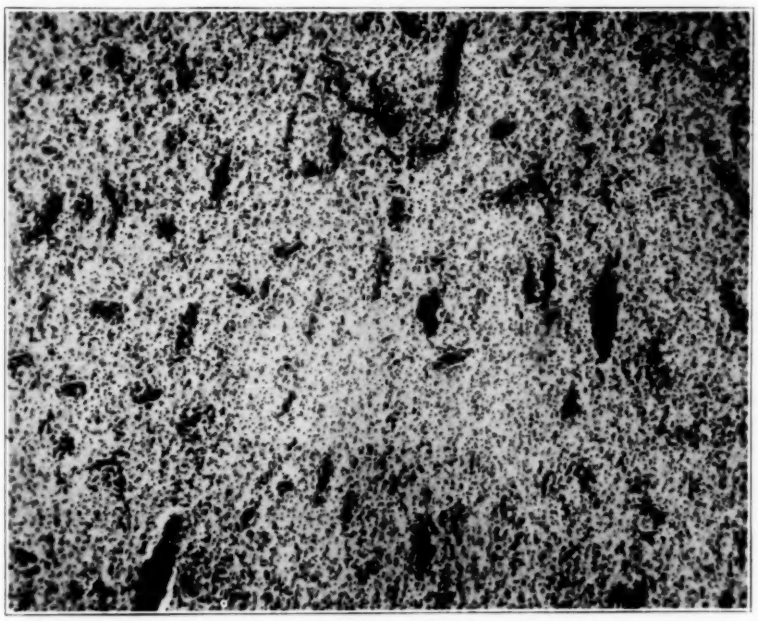

Fig. 10.—Occipital lobe. The dense rings are perivascular spaces packed with gitter cells, a few hematogenous elements and basophil metachromatic substances. Toluidin blue; × 60.

As a rule, inflammation is foreign to this disease; I have never seen it in my cases, including the one here recorded, though the similarity, with some staining methods, may be great.

1. Alzheimer, A.: Beiträge zur Kenntniss der pathologischen Neuroglia und ihren Beziehungen zu den Abbauvorgängen im Nervengewebe, Nissl-Alzheimer's Arb. **3**:493, 1910.

2. Kraus, W. M.; Lhermitte, J. J., and Bertillon, F.: Mucinlike Bodies in the Central Nervous System of Epidemic Encephalitis, Arch. Neurol. & Psychiat. **12**:620 (Dec.) 1924.

3. Bailey, P., and Schaltenbrand, S.: Die muköse Degeneration der Oligodendroglia, Deutsche Ztschr. f. Nervenh. **97**:231, 1927.

mucous degeneration.

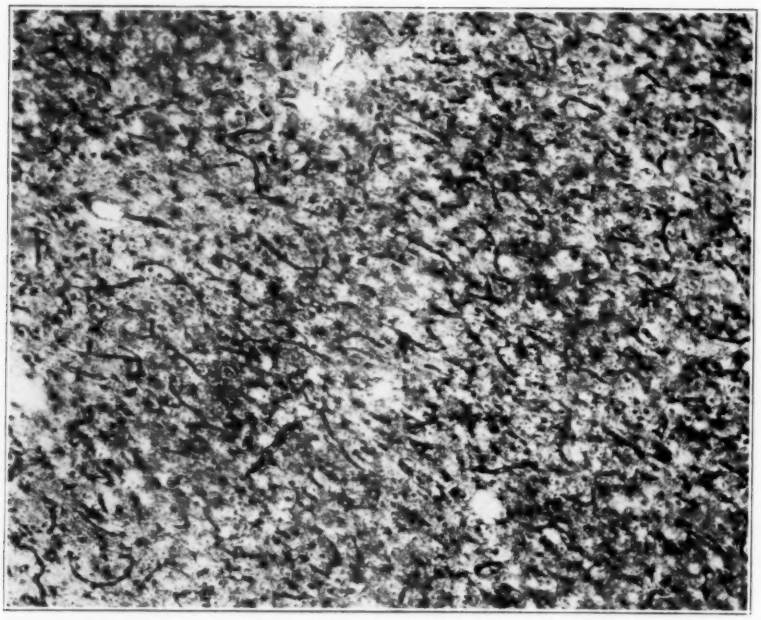

Fig. 11.—Optic thalamus. Immense vascularization. Cajal's gold corrosive sublimate stain; × 95.

Fig. 12.—Optic thalamus. Cavities and other changes described in text. Same stain as in fig. 11; × 27.

Frequently the blood vessels were proliferated and newly formed, especially in the cerebellum and the optic thalami, in which their number may be immense (fig. 11). In the optic thalami, the excessive vascularization was associated with the formation of cavities or lacunae (fig. 12) lined by remnants of nerve tissue and surrounded by proliferated glia. These lacunar conditions of degenerations denote a far advanced parenchymal destruction, which in the optic thalami was even more advanced than in the occipital and frontal lobes or the cerebellum — parts which, as noted, were particularly affected.

The pia-arachnoid was hyperplastic; its meshes were dilated (fig. 13) and enclosed a large number of various cell elements such as lymphocytes, mesothelial cells, fibroblasts, gitter cells and macrophages filled with pigment.

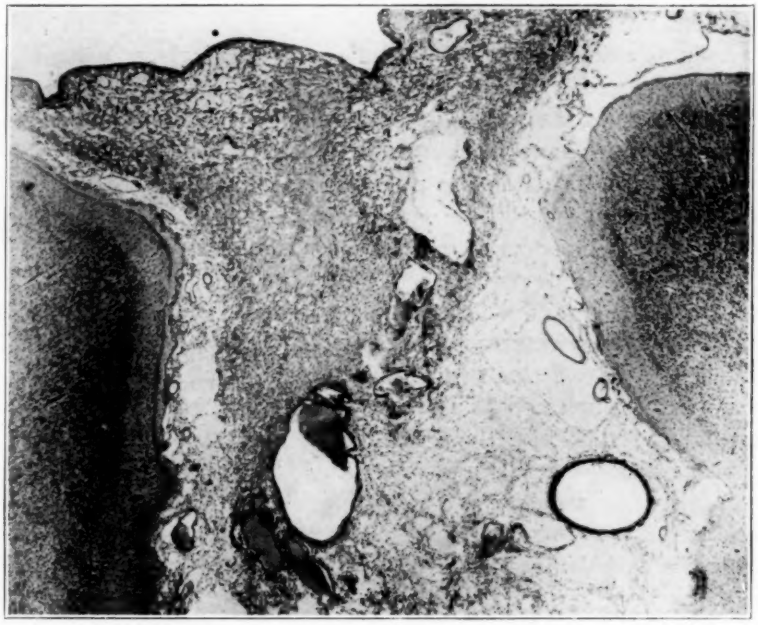

Fig. 13.—The arachnoid membrane stretching over a sulcus filled with a hyperplastic pia. Toluidin blue; × 16.

These have been more fully described elsewhere.[4] The same may be said of the choroid plexus in which the tuft cells were large and vacuolated, often resembling gitter cells.

GENERAL SUMMARY

Widespread parenchymatous degeneration and atrophy of the hemispheres, optic thalami and some portions of the cerebellum; diffuse changes in the ganglion cells typical of amaurotic family idiocy and hyperplastic or fibrous leptomengitis with hyperplasia of the blood vessels and lacunar degeneration of the optic thalami were present.

4. Hassin, G. B.: A Study of the Histopathology of Amaurotic Family Idiocy (Infantile Type of Tay-Sachs), Arch. Neurol. & Psychiat. **12**:640 (Dec.) 1924.

COMMENT

The parenchymatous changes were the primary phenomena; the mesodermal (vascular and pia arachnoid) changes were secondary. As pointed out elsewhere,[5] the pia arachnoid forming the subarachnoid space receives the waste products from the brain tissues, in this case, as in many others, enclosed within macrophages and gitter cells. Their presence in the pia-arachnoid provoked numerous reactive phenomena that resulted in the histologic picture of a fibrous leptomeningitis. The changes in the blood vessels, in the form of distended perivascular spaces, excessive proliferation and new formation, were also a manifestation of severe parenchymatous lesions. These were in evidence even macroscopically as a diffuse atrophy. Of the atrophied regions, I should like to mention especially the optic thalami. These structures were reduced to mere remnants, the remaining parts showing a degeneration that was far advanced, few ganglion cells and nerve fibers, enormous vascularization and formation of lacunae. In specimens stained with the silver method of Bielschowsky, the condition resembled that of the corpus callosum (fig. 8) and has been fully described elsewhere.[4]

The view that one of us (G. B. H.) expressed in previous publications,[6] that in amaurotic family idiocy the destruction of the optic thalami is the outstanding feature, is also sustained by the present case. "No normal cells" were found in the thalamus by Grinker in his two cases: "many were completely disintegrated, and neuroglial proliferation was present."[7] The claim of Leiner and Goodhart[8] that in their two cases the optic thalami were "practically" normal is evidently a misunderstanding, for in amaurotic family idiocy nothing is "practically" normal; everything is abnormal. Their assertion that the observations of one of us (G. B. H.) in the optic thalami do not correspond with the facts and require a different explanation, which they try to give, is difficult to understand. Their photomicrographs are unconvincing; it is possible that by some accident the optic thalami were studied superficially or were not studied at all.

Of equally great interest is the marked atrophy of the hemispheres and the cerebellum. While early students of this disease, such as

5. Hassin, G. B.: Notes on the Nature and Origin of the Cerebrospinal Fluid, J. Nerv. & Ment. Dis. **59**:113, 1924; Effect of Organic Brain and Spinal Cord Changes on Subarachnoid Space, Choroid Plexus and Cerebrospinal Fluid, Arch. Neurol. & Psychiat. **14**:468 (Oct.) 1925.

6. Hassin, G. B.: A Case of Amaurotic Idiocy, Late Infantile Type (Bielschowsky) with Clinical Picture of Decerebrate Rigidity, Arch. Neurol. & Psychiat. **16**:708 (Dec.) 1926. See also footnote 4.

7. Grinker, R. R.: The Microscopic Anatomy of Infantile Amaurotic Idiocy, Arch. Path. **3**:768 (May) 1927.

8. Leiner, Joshua; and Goodhart, Philip: The Infantile Type of Family Amaurotic Idiocy, Arch. Neurol. & Psychiat. **17**:616 (May) 1927.

B. Sachs and Karl Schaffer, were inclined to the opinion that in amaurotic family idiocy the brain and the spinal cord were devoid of any macroscopic or gross signs, lately a number of gross changes have been described which Schaffer [9] considered characteristic. He included among other changes the gaping sylvian fissure, the abnormal fissuration of the occipital lobe, the abnormal architecture of the central convolutions, the presence of a dorsal lateral fissure in the spinal cord and some other features. He even goes so far as to consider the foregoing "abnormalities" as signs of an abnormal or retarded development of a brain that is inferior to a human brain and resembles that of an ape. In short, the gross changes referred to are a manifestation of atavism, a view not sustained by Bielschowsky,[10] another student of this disease. Whether the foregoing gross changes are atavistic phenomena or as is more probable, the result of an atrophy that is far advanced, the latter assumption may help in many cases to diagnose amaurotic family idiocy macroscopically. In fact, it is hard to point out any other condition, not excepting juvenile general paralysis, in which a brain would exhibit so far advanced an atrophy as is seen in some cases of amaurotic family idiocy, especially the cases with a protracted course. Such cases, usually classified as the late infantile type, show diffuse atrophy of the cerebellum, a late onset at the age of 3½, a prolonged duration (four years), and other features that have been discussed elsewhere.[6] In the case here recorded the cerebellar atrophy was not diffuse but partial; the onset occurred when the child was about 2½ years, and the entire duration was three years and nine months; hence, this case might be considered a transitional form between the infantile and the late infantile forms. One might assume that the onset was much earlier and that the child's mental condition was overlooked. This would seem unlikely, for the child had been under medical supervision (for spinal trouble?) since the age of 3 months and, as said previously, did not reveal alarming signs or symptoms for about two and one-half years. As the child died at the age of 3 years and 9 months, the actual duration of the malady was fifteen months. However, the advanced destruction of the brain would suggest a much earlier onset—in spite of the absence of corresponding clinical features—and a course much more protracted. In the infantile form of amaurotic family idiocy, this is usually eighteen months, though a few cases have been recorded in

9. Schaffer, Karl: Tatsächliches u. hypothetisches aus der Histopathologie d. infantile-amaurotischen Idiotie, Arch. f. Psychiat. **64**:570, 1922; Ueber das morphologische Wesen und die Histopathologie der hereditaersystematischen Nervenkrankheiten, Berlin, Julius Springer, 1926.

10. Bielschowsky, Max: Zur Histopathologie und Pathogenese der amaurotischen Idiotie mit besonderer Berücksichtigung der zerebellaren Veränderungen, J. f. Psychol. u. Neurol. **26**:123, 1920.

which the course was much longer. In Koller's [11] case, the duration was four years; in Mühlberger's,[12] three years and nine months; in Wolfsohn's [13] two years and three months; in Schaffer's,[8] two years and four months, and in Ostertag's,[14] three years and two months. The cases of Koller and Mühlberger were not studied histologically; they did not show the cherry-red spot, which was also absent in our case. Absence of a cherry-red spot, therefore, should not deter one from making a diagnosis of amaurotic family idiocy (Tay-Sachs type). Instead of a cherry-red spot, other changes may be found in the fundi which might be looked on as equivalents. In our case, for instance, the fundi showed grayish discoloration of the retina and a nebulous appearance; in Koller's case, there "was in the region of the macula a slight, veil-like, milky-bluish haze gradually fading into the color of the surrounding retina." In Mühlberger's case, the macular region was reddish, surrounded by a dark pigment ring.

Insignificant as such ocular manifestations might seem as compared with the cherry-red spot, in the presence of other signs and symptoms, they may help to make a correct diagnosis, for in the absence of the characteristic spot a diagnosis of amaurotic family idiocy is generally not considered safe.

CONCLUSIONS

1. Amaurotic family idiocy, the infantile type of Tay-Sachs, may have an insidious onset—of more than two years—and may not show any alarming symptoms for a considerable period of time.

2. The course may also be protracted, lasting up to four years.

3. Characteristic changes in the fundi, such as the cherry-red spot, may be absent in this disease.

4. The cherry-red spot may be replaced by other changes less typical, but important enough to help in making a correct diagnosis.

5. The cerebral atrophy, especially in cases with a prolonged course, may be so marked that a diagnosis of amaurotic family idiocy should be seriously considered.

6. In protracted cases, the optic thalami are also more atrophied than any other part of the brain.

11. Koller, Carl: Two Cases of "A Rare Fatal Disease of Infancy, with Symmetrical Changes in the Macula Lutea" (Kindon), M. Rec. **50**:266 (Aug. 22) 1896.

12. Mühlberger, A.: Die familiäre amaurotische Idiotie u. ihre Diagnose, München. med. Wchnschr. **50**:1969 (Nov. 10) 1903.

13. Wolfsohn, J. H.: Amaurotic Idiocy; Pathologic Report by J. R. Oliver, Arch. Int. Med. **16**:257 (Aug.) 1915.

14. Ostertag, B.: Entwickelungstörungen des Gehirns und zur Histologie u. Pathogenese, besonders der degenerativen Markenkrankung bei amaurotischer Idiotie, Arch. f. Psychiat. **75**:355, 1925.

A BREAST AND BOTTLE SHY INFANT

NURSING ONLY WHEN ASLEEP *

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PITTSBURGH

The following case of breast and bottle shyness in an infant is reported on account of two unusual features; first, cases in which infants are both breast and bottle shy are rare; second, the fact that this infant would nurse from the bottle in the dark only, and even then never unless it was given the bottle while asleep.

REVIEW OF THE LITERATURE

Cases of breast shyness alone are rare and such authorities as Sedgwick and Cole [1] say that although they have never seen such cases, authentic reports have been made of children who refuse to nurse, turn their heads away, cry lustily and appear to be afraid of the breast. When such children are blindfolded or nursed in a dark room, it is said that they nurse like any other child. Whether or not this is a condition of fear in the infant, or a habit based on a neuropathic diathesis formed by improper technic in nursing during the first days of life, is a matter of considerable doubt.

Von Reuss [2] says:

> Just as the premature child is frequently incapable of nursing the breast, so there are also full-term infants who are decidedly weak or lazy nursers. These infants do not grasp the nipple or allow it to drop after a few weak suckling motions or go to sleep. Often these symptoms disappear after the first or second day of nursing, so that it is difficult to decide whether actual weakness or minimum needs underly. Often, however, in spite of all efforts, days pass without success. Here we are, as a rule, dealing with a class of small, delicate newborns with low birth weight, for whom no satisfactory designation exists. They are neither debilitated nor are they constitutionally abnormal; they can respond fully to the demands of extra-uterine life. Perhaps they gain more slowly, and perhaps regain their birth weight later. During the first months they may appear delicate, but regain the deficit in the second half year and develop further as normal children. Their whole conduct reminds one of healthy premature infants and, indeed, many cases may be undiagnosticated premature babies; though certainly not all are such. In the latter case, where the infant is full-term, but shows characteristics of the premature child, we must presuppose an abnormal functional retardation. In rarer cases, there is a peculiar aversion to the breast on the

* Received for publication, July 30, 1927.

1. Sedgwick, J. P., and Cole, W. C. C., in Abt, I. A.: Pediatrics, Philadelphia, W. B. Saunders Company, 1924, vol. 2, p. 608.

2. Von Reuss, August Ritter: Die Krankheiten des Neugeborenen, Berlin, 1914, p. 99.

part of the infant. The infant does not give the impression of being a weak nurser or of having a lack of appetite and there is no fault in the mother's breast. But as often as one puts the infant to the breast, it starts to cry, turns its head to one side, tugs at the nipple, and twists itself, so that it is not possible to get the child to nurse. Most of these breast shy infants will nurse well from the bottle. In these cases, it is a mistake to fully satisfy the nutritional requirements. These infants can only be forced to nurse by hunger.

Finkelstein [3] believes the true cause to be, not weakness or laziness, but awkward nursing due to an imperfect muscular sense; he does not agree with Birk, who believes that these cases are the result of minor cerebral birth injury, pointing to the fact that these disturbances of nursing continue many weeks in infants otherwise in good health. He classifies the real breast shy infant as a constitutional neuropath, and to this class the child whose case is here reported undoubtedly belongs. In these cases, he believes that there is a disturbance (von Reuss states that there is a congenital absence) of the sucking reflexes. In some cases, the nursing reflex center in the mouth is further back than normally, and the condition may be corrected by the use of an extra long nipple. In other cases, this center is entirely absent, and the infant must be fed by a tube for some time, until it gradually learns to nurse properly, which may take a long time. Here one has to do with delayed development, but in other cases one has to deal with an aplasia of the nervous pathways or centers. In one of Finkelstein's cases, failure of development of the nucleus was proved by a double congenital ptosis, oculomotor and facial paralysis. More commonly the disturbance is due to idiocy. In congenital athyreosis and Mongolian idiocy, nursing difficulties are the rule, so that they can be considered a diagnostic symptom of these conditions. Idiocy, however, is not necessarily present in these cases. Finkelstein recalls a case of practically complete absence of the reflexes up to the sixth month; later the intelligence developed normally. Occasionally the swallowing reflex is also affected by paralyses in cerebral or bulbar disease or by convulsions.

Czerny and Keller [4] call attention to the receding lower jaw (prognathie) as a cause of difficult nursing; they claim this to be an indication of pathologic changes in the base of the skull. According to von Basch,[5] the center for nursing in animals is in the medulla oblongata, which would explain why infants with hydrocephalus, microcephalus and even anencephalus can nurse.

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3. Finkelstein, H.: Lehrbuch der Säuglingskrankheiten, ed. 3, Berlin, Julius Springer, 1924, p. 70.

4. Czerny, A., and Keller, A.: Des Kindes Ernährung, Ernährungsstörungen und Ernährungstherapie, Vienna, Franz Deuticke, 1923, vol. 1, part 1, p. 32.

5. Von Basch: Jahrb. f. Kinderh. **38**:68, 1894.

There is, of course, a group of children who cannot nurse on account of obstacles such as deformities or ulcers of the mouth or lips; macroglossia, cleft palate, congenital atresia of the nose or other nasal obstructions, prematurity, cerebral hemorrhage and various local injuries at birth. Some cannot nurse because the mother's nipples are retracted or are too small or too large, the breasts are slow-going or extremely tense, the milk appears late or the technic is faulty in nursing. These cases do not, of course, properly belong to the group of cases of breast or bottle shy infants in whom such causes must be eliminated.

As has been pointed out by Hector Cameron,[6] negativism in the neuropathic child may develop at an extremely early age. The sudden transition from intra-uterine life, where no sound or light reaches the infant, to the reflex stimuli of postuterine life reacts markedly on the neuropathic child. Cameron believes that the most common cause of shyness in nursing in this type of infant is the nervous unrest of that reflex act of nursing which works so well in the placid and quiet child. He recommends that these infants be placed to the breast when they are drowsy and sleepy, not when they are crying furiously, and that they be given chloral and bromides to induce drowsiness. When this has acted, and the infant is sleeping soundly, it should be carried gently to its mother and the nipple inserted. These children usually show hypertonicity of the muscular system.

One occasionally sees cases of obstinate refusal to take more solid food, like farina or zweiback, in older children; these children have usually been bottle fed too long, and they greedily take fluid food. Attempts to make them take solid food result in paroxysms of rage, pushing away of the food, choking and gagging, even going to the verge of convulsions. If the food is taken, rumination may occur. The most experienced of children's nurses will fail at first; only strict perseverance and repeated attempts finally succeed. Such children have been known to starve to death.

REPORT OF A CASE

History.—R. L., a white girl, a full term first baby, Jewish, born on Feb. 18, 1926, was first seen on April 12, 1926, at the age of 8 weeks. The mother, aged 31, apparently was robust and in good health; the father, aged 35, was in good health. The mother had had the usual nausea of pregnancy during the first eight or nine weeks. She had not had any miscarriages; the Wassermann test was negative. There was no history of consanguinity in the parents and no history of similar cases in either the mother's or the father's family. There was no history of organic nervous diseases, but there was a neuropathic tendency.

The infant was delivered with forceps at the Magee Hospital by Dr. Harold A. Miller. There was asphyxia livida, the respirations were delayed thirty

6. Cameron, Hector: The Nervous Child, London, 1924.

seconds, and Schulze-Ahlfeld resuscitation was necessary. The diameters of the head were: suboccipitobregmatic, 9.5 cm.; suboccipitofrontal, 10 cm.; occipitofrontal, 10 cm.; occipitomental, 15 cm.; bitemporal, 8 cm. and biparietal, 9.5 cm. The circumferences of the head were: suboccipitobregmatic, 30.5 cm.; suboccipitofrontal, 31.5 cm.; occipitofrontal, 34.5 cm. and biscapular, 36 cm. The length of the head was 52.5 cm.

The mother's breasts filled well by the third day. From the beginning, the infant refused to take the breast. It took ½ and 1½ drachms (1.8 and 4.6 cc.) of a formula from the bottle, the second and third days, then refused the bottle also and had to be fed by tube. A premature feeder was tried, but the infant could not be fed by this means. There was occasional vomiting during the first week. The birth weight was 3,200 Gm., and on the fifth day it fell to 2,935 Gm., after which there was a slow rise. A formula of skimmed milk, 6 ounces (178 cc.) milk sugar, 3 level tablespoons, water 12 ounces (355 cc.) and lactic acid 30 minims (1.9 cc.), brought to a boil, was given at this time. The only rise of temperature noted was on the fifth day, when it was 100.8 F. The stools, from four to eight daily, were greenish and curdy. There was slight vaginal bleeding on the fourth day.

So far as could be ascertained, there was no error in the technic of feeding. The infant did not make any effort to take the breast at the first attempt at nursing; it could not be forced to take the nipple in its mouth. Later, it shook its head when the nipple was forcibly placed in its mouth. The mother's breasts were pumped by the Abt Electric Breast Pump, which was used for several months, and the milk thus obtained was given to the infant by the tube and later by the spoon. Later a formula of skimmed boiled milk was given when the breast milk was not sufficient in quantity. After the third week, it was found that if the infant was given the bottle when asleep and in a darkened room, from 1 to 1½ ounces (29.7 to 44.55 cc.) of milk could be given from the bottle. Later, in the same way, as much as 3½ to 4 ounces (104 to 118 cc.) would be taken if the child was fast asleep; if it awakened, it would stop feeding and would not take any more until it was asleep again. At 4 weeks of age, the child began to vomit after feedings, which at this time were chiefly spoon feedings. The vomiting was not projectile, and there was no visible peristalsis or tumor. The stomach was washed with a 5 per cent solution of sodium bicarbonate, and one teaspoonful of farina was given in 1 ounce of whey before each feeding, with good results. Then skimmed milk with farina water and cane sugar was given as supplementary feeding, and this was well retained. Gravity cream was gradually added to this formula. Feeding with thick farina was tried in order to give as many calories as possible in small bulk, but it was not well taken. Diminishing the number of feedings to three daily and even starvation for twenty-four hours did not improve the infant's appetite or modify its stubborn refusal to nurse. The child has always been inclined to drowsiness, especially up to the fifth month, and this tendency persists to the present. At 6 months, she began to take more solid food, such as farina and vegetables, but she would never take zweiback. There was some difficulty in giving solid food at first, but at present, it is well taken.

She held her head up at, or before, the fourth month; followed the finger or a toy at the fourth month, and sat up at the sixth month. The first tooth erupted at the eighth month; she walked if supported at 1 year of age, said "da-da" and "ma-ma" at 8 months, and at 1 year says three words consecutively. It was noted on the first day of life that the infant cried more than usual. At 1

year of age, it cried on seeing strangers, and had a hyperacute reaction to noises and strong lights. In general, she has developed much more rapidly mentally than physically.

Physical Examination.—(April 12, 1926.)—The infant was poorly developed, and she reacted strongly to slight noises or bright lights. The fontanel was not bulging, and craniotabes was not present. The child did not have stiffness or retraction of the neck. The pupils reacted normally. Examination did not reveal nystagmus or strabismus or any abnormalities of the face, head, mouth, lips, throat or nasal structures. The infant had a geographical tongue. The heart and lungs did not show any abnormalities and the thymus was not enlarged. The abdomen was distended, and umbilical hernia was present. The liver and spleen were not enlarged. The knee reflexes were exaggerated; the Kernig and Brudzinski sign were not present. There was general muscular hypertonicity. Cranial nerve or other paralysis was not present. A no. 14 F stomach tube was passed without difficulty, and gastric contents were not retained. She passed four or five stools daily; occasionally they were greenish with fine curds and much mucus but no blood.

At 1 year of age, the weight was 16 pounds and 7 ounces (7,460 Gm.); the head circumference was 44 cm.; bitemporal diameter, 24 cm.; sagittal occipital frontal diameter, 28 cm. The mentality was that of the average 1 year old child.

TREATMENT

In the treatment of such infants, the proper method is to feed pumped or expressed breast milk if obtainable from the bottle or spoon if possible; if necessary, it should be fed by tube, as in the present case. In order to maintain the breast secretion, the Abt Electric Breast Pump is indispensable.

In this way, underfeeding is prevented as far as possible, and if necessary, supplementary feedings are given. A certain degree of underfeeding is of advantage in maintaining the infant's appetite. One should put the infant to the breast regularly, or at least attempt it, and afterwards give the expressed milk in small measured doses, even if the milk is abundant. In the majority of cases, the infant begins to nurse after several days, especially when the breast milk becomes freer and the milk more easily obtained from the breast. The conditions are less favorable when none or very little milk can be obtained from the mother's breast. In these cases, a wetnurse should be secured. Schutz has advised putting the nipple in the infant's mouth and squeezing the stream into it. The sense of taste in the young covers a relatively much greater area of mucosa than in older persons, and the appetite may be stimulated by the spray of milk on the hard and soft palate, the mucosa of the cheeks and pharynx. After the attempt at nursing which requires much patience and perseverance on the part of the mother and the attendants, one should offer the child only water or tea for a time and not too much milk. Correct technic of nursing, and the proper preparation of the formula, if one is used, are essential in the treatment of breast or bottle shy infants and in the prevention of the development of this trait. A

thorough and careful examination of the mother and infant for the detection of any possible faults or abnormalities must be made in all cases presenting this type of difficult feeding. A congenital absence of the sucking reflex must be thought of, especially in the type of case here described. Oui describes a case in which an infant nursed only when an extra long nipple was used. In this case it was thought that the center for nursing was unusually far back. Cameron's suggestion of giving chloral bromides to produce drowsiness would hardly be practical in long drawn out cases.

Limiting the number of feedings to three daily or even starvation for a day or two may be tried, but these measures are usually unsuccessful in getting these infants to nurse; and, since many of them are badly undernourished, and on account of their tender age, this treatment is not without danger. A change to a wetnurse does not succeed, except when there is some abnormality of the mother's breast. If the condition persists as the infant gets older, attempts should be made to feed more concentrated foods, such as thick farina, after Sauer's method, in order to give a greater number of calories in smaller bulk. When such feedings are given, care should be taken to provide an adequate water intake. On account of its sweetness, cane sugar may be of service in preparing the formulas that the infant will take.

SUMMARY

A case of breast and bottle shyness in an infant who showed the unusual peculiarity of taking the bottle only in the dark and when asleep, is presented.

This type of infant must be classified as a constitutional neuropathic child with either a disturbance or congenital absence of the sucking reflex.

In these cases, a faulty nursing technic and faults or abnormalities on the part of mother and infant are the factors for which one must search carefully; if found, they must be eliminated.

The treatment in these cases requires much patience. If possible, the mother's breast milk should be maintained by means of the electric breast pump and fed by tube or spoon. Formulas containing high calories in small bulk, such as lactic acid milk or, especially in cases with vomiting, thick farina gruel, are advisable. Overfeeding should be avoided. Feeding by tube is often required for some time.

CONGENITAL OCCLUSION OF DUODENUM BY A COMPLETE DIAPHRAGM*

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My interest in congenital occlusion of the duodenum by a complete diaphragm was aroused by the following case:

REPORT OF A CASE

History.—On December 27, 1924, a child, 4 days old, was seen at its home, and a history of normal labor and the delivery of an apparently normal child was obtained. However, when breast milk or water was given, it was vomited, usually within an hour, the vomitus being of a dark greenish color. A very small amount of dark colored mucus had been passed by rectum. Urine had not been voided.

Examination.—Physical examination showed an emaciated child lying in the dorsal position, with mouth wide open and staring eyes; the skin and mucous membranes were markedly icteric; the pupils were equal, and reacted to light. The heart and lungs appeared to be normal. The upper part of the abdomen was tympanitic, but not distended, and the lower part was flat. A moderate degree of talipes calcaneous was present. Twice during the examination, the child vomited a bile stained liquid without fecal odor.

Treatment.—The child was admitted to St. Vincent's Charity Hospital with a diagnosis of congenital intestinal obstruction. An intraperitoneal saline infusion was given to relieve dehydration, but operative interference was not deemed advisable as the child was moribund. The bladder was catheterized, but urine was not obtained. An obstruction was not felt in the rectum. An enema gave a nearly clear return. Expressed breast milk and casein milk were given, but were vomited; on one occasion the food was retained for four hours. The rectal temperature rose to 104.6 F., then dropped to 101.8 F. just before death, which occurred thirteen hours after admission and when the child was 5 days of age.

Autopsy.—On opening the abdomen, the stomach and duodenum were found distended, the latter to about two thirds of the size of the former, from which it was separated by a pylorus of normal diameter. Pressure on the duodenum caused it to pouch in its lower end, but its contents did not enter the jejunum. On opening the stomach and duodenum, a well marked pyloric ring was found. The stomach and duodenum contained bile and mucus. The common duct was patent, bile being easily expressed through it. Four centimeters below the pyloric ring, and 1.5 centimeters below the papilla of Vater, a complete transverse membranous septum or diaphragm occluded the duodenum. Neither air nor water could be forced past it. The rest of the small intestine and all of the large intestine were patent but collapsed, and of a "worm-like" appearance. Further obstructions of any character were not encountered. The bladder was small and did not contain any urine.

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REVIEW OF THE LITERATURE

Calder is credited by Davis and Poynter [1] with having recorded in the *Journal of Medical Essays,* Edinburgh, 1733, the first case of a congenital duodenal occlusion. Only a comparatively few cases were reported up to 1901, for, in that year, Cordes,[2] of New York, was able to gather data on only fifty-seven cases. Spriggs,[3] in a comprehensive article on "Congenital Intestinal Occlusion" in *Guy's Hospital Reports* of 1912, listed ninety-two cases in which the defect occurred in the duodenum. To these Cautley,[4] in 1919, added eleven cases, but rejected three of Spriggs' cases as doubtful. A search of the available literature, from 1919 to 1926 inclusive, has revealed twelve more cases of this nature (French,[5] 1; Jewesbury,[6], 2; Schroeder,[7] 2; Smellie,[8] 2; Marfan,[9] 1; Ralston,[10] 1; Seidlin,[11] 1; Maitland,[12] 1; Magee,[13] 1), making a total of 113 cases including the one here reported.

The site of the defect in these cases is as follows:

Above the biliary papilla	34
Opposite the biliary papilla	19
Below the biliary papilla	25
At the duodenojejunal flexure	19
Unclassified	16
Total	113

The ninety-two cases reported by Spriggs [3] constituted 28 per cent of the total number of congenital occlusions of the entire intestinal canal,

1. Davis, D. L., and Poynter, C. W. M.: Congenital Occlusions of the Intestines, Surg. Gynec. & Obst. **34**:35, 1922.

2. Cordes, L.: Congenital Occlusion of the Duodenum, Arch. Pediat. **18**:401 (June) 1901.

3. Spriggs, N. I.: Congenital Intestinal Occlusion, Guy's Hosp. Rep. **16**:143, 1912.

4. Cautley, E.: Duodenal Stenosis, Brit. J. Child. Dis. **16**:65, 1919.

5. French, H. L.: Congenital Atresia of Duodenum with Report of Case, J. Michigan M. Soc. **24**:240 (May) 1925.

6. Jewesbury, R. C.: Congenital Stenosis of Duodenum, Proc Roy. Soc. Med. **16**:10, 1922-1923.

7. Schroeder, C. H.: Diag. of Congenital Duodenal Occlusion, J. A. M. A. **82**:686 (March 1) 1924.

8. Smellie, J. M.: Three Cases of Congenital Duodenal Occlusion, Brit. J. Child. Dis. **21**:192 (July-Sept.) 1924.

9. Marfan et al.: Congenital Stenosis of Duodenum, Nourrisson, **12**:101 (March) 1924.

10. Ralston, G.: Congenital Occlusion of First Part of Duodenum, Brit. M. J. **2**:1005 (Nov.) 1925.

11. Seidlin, S. M.: Congenital Duodenal Septum with Obstruction, Bull. Johns Hopkins Hosp. **37**:328 (Nov.) 1925.

12. Maitland, C. D.: Congenital Duodenal Atresia, Brit. M. J. **2**:609 (Oct.) 1925.

13. Magee, A. V.: Congenital Occlusion of Duodenum, Brit. M. J. **1**:826 (May) 1926.

excluding those at or near the anus. Davis and Poynter,[1] in 1922, collected 392 cases of congenital occlusion of the intestines between the pylorus and anus, and listed 134 or one third of these as being in the duodenum, 59 above and 75 below the papilla. This would increase the total of reported cases considerably, but as these authors do not refer in their bibliography to anything published since 1916, and do not give references for each newly reported case, I have decided to add my cases to the Spriggs-Cautley collection.

CHARACTERISTICS OF THE CONDITION AND TREATMENT

The occlusion of the intestine may be partial or complete. To the former belong cases of simple narrowing of the bowel and cases of perforated diaphragm. Complete occlusion occurs by annular constriction which entirely obliterates the lumen; by a complete membraneous diaphragm; and by an atresia of a portion of the intestine, the upper segment ending in a cul-de-sac, and either being united to the lower segment by a short band or by a longer thread-like band, or having no connection with it.

It has been impossible to determine with exactness the number of cases in which the diaphragm was complete. Ballantyne [14] listed eleven cases of congenital septal occlusion reported between 1856 and 1900; and five of the thirteen cases I have collected, including my own observation, were of this nature. The total number of cases to date probably does not exceed thirty-five or forty.

Many explanations have been advanced as to the etiology of this malformation, but no one explanation seems to account for all types of malformations in all locations. Bland Sutton [15] maintains that the malformation takes place at the site of an "embryological event" such as occurs in the duodenum, where, in the fourth week of fetal life, the liver and pancreas develop as buds from its wall. A plausible hypothesis is that of Tandler,[16] who states that the duodenum near the papilla of Vater is occluded during the second month of fetal life by epithelial proliferation, which normally undergoes canalization at a later date. Partial or complete failure of this canalization is his explanation of the partial or complete occlusions of the intestine. The reader is referred to an excellent article by Pehu and Augerge [17] for a list of other suggested causes.

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14. Ballantyne, J. W.: Manual of Antenatal Pathology and Hygiene, New York, William Wood & Company, vol. 2, p. 532, 1905.

15. Sutton, J. Bland: Imperforate Ileum, Am. J. M. Sc. **98**:457, 1889.

16. Tandler, J.: Zur Entwickelungsgeschichte des menschlichen Duodenums in frühen Embryonalstadien, Morphol. Jahrb. **29**:187, 1900.

17. Péhu, M., et Auberge, A.: Rétrecissements congenitaux du duodénum, Arch. d. méd. d. enf. **27**:330, 1924.

The symptoms are those of high intestinal obstruction. Vomiting occurs in every case whether or not food or water are given by mouth; it is forceful, and, as would be expected, begins early, usually by the second day. Except when the obstruction lies above the openings of the bile ducts, the vomitus contains bile, but hematemesis is less frequent. Constipation is absolute, and inanition and loss of weight are rapidly progressive. By contrast with the flat lower half of the abdomen, the epigastrium appears to bulge; sometimes a left to right peristalsis is visible.

When a diagnosis is made, congenital pyloric stenosis must be considered, but should be easily excluded. Vomiting in this condition is absent during the first days of life; the vomitus does not contain bile, and the child usually has an initial gain in weight with normal stools. Rectal examination and enemas help in differentiation from imperforate anus and rectal occlusions. The roentgen ray enables one to locate the site of the obstruction, whether it is esophageal, gastric or intestinal, and to determine its completeness.

The prognosis is not hopeless, in spite of the fact that recovery has not occurred in cases of complete obstruction. Ernst,[18] of Copenhagen, in 1916, reported the first successful operation for a congenital duodenal occlusion, his patient being a 10 day old boy with a duodenal stenosis cured by a duodeno-entero anastomosis. Grulee[19] stated that four patients with congenital intestinal occlusion had been successfully operated on up to 1926.

The treatment is, of course, surgical, and its success demands an early diagnosis. The possibility of this malformation must therefore be kept in mind. Whatever surgical means are employed to circumvent a complete obstruction, Cloggs'[20] suggestion that the collapsed intestine below the obstruction should be gently inflated by water or air should be carried out. This has been successfully done, and it not only makes the anastomosis easier to perform, but this procedure shows whether or not any further obstructions are present.

SUMMARY

1. Congenital occlusions of the duodenum by a complete membranous diaphragm are rare.

2. They constitute from one-quarter to one-third of all congenital occlusions of the duodenum, of which about 113 have been reported.

3. Early diagnosis and operation offer the only hope of cure.

4. A new case of this condition is reported.

18. Ernst, N. P.: A Case of Congenital Atresia of the Duodenum Treated Successfully by Operation, Brit. M. J. **1**:644, 1916.

19. Grulee, C. G.: Clinical Pediatrics, New York, D. Appleton & Company, 1926, vol. 3, p. 151.

20. Clogg, H. S.: Congenital Intestinal Atresia, Lancet **2**:1770, 1904.

Progress in Pediatrics

PHYSICAL PROBLEMS OF ABNORMAL BEHAVIOR*

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Conduct is an interaction between the constituent elements of human nature and the natural and social environment. Behavior is personal conduct, or conduct in its external relations. While conduct may be defined briefly as the sum of the reactions of an organism in and through living, behavior implies action in the presence of others. Behavior essentially, therefore, possesses a social quality which need not be existent in conduct, although Dewey regards conduct as "A social reaction in and because of environment."

Whether behavior is regarded as action or reaction, it is patent that it must depend on at least two elements dealing with human organization. Behavior must be viewed in terms of structure and function. Abstract definitions are inadequate to indicate the complicated interrelations between man's internal mechanisms and the external forces which surround him. Internal, as thus used, practically means inherent.

Man's behavior is conditioned by the state of development of his nervous system, but no less so by the condition of his visceral, skeletal and reproductive systems. Man is not, however, an assemblage of differentiated systems. Massing together respiratory, circulatory and excretory systems with cerebrospinal and sympathetic nervous systems does not constitute man as a functioning unit. Hence, structure, while vitally important to behavior, is less consequential than function. Obviously, a man cannot use a hand if it has been amputated, but he may possess the hand and be unable to utilize it. The function of the hand is of greater importance than the mere esthetics of its presence. For example, many a person with a paralyzed, useless hand is hampered in behavior to a far greater extent than he would be if the hand were to be amputated and an artificial hand applied.

The existence of a heart with its regularity of contraction is essential, but just as the actuary appreciates the influence of its function on life expectancy, so may one grasp the influence of impaired cardiac function on behavior. The dire effects of palpitation, dyspnea, arrhythmia and

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edema are not limited to the circulatory system, but enter into the complete reaction systems of the individual.

Strabismus and myopia involve structural alterations, but their consequences depend on the degree of interference with function. The irregularities of behavior of the child with unrecognized myopia may soon be relieved by the correction of the functional deficiency. This suffices to indicate that behavior presents dual physical problems relating to the structure and function of various reacting systems which, however, must be united and integrated in action in order to conserve or advance man's harmony in his environment.

ABNORMAL BEHAVIOR

What is meant by abnormal behavior? What does the word abnormal mean? How is abnormal to be differentiated from normal behavior? Only the same mechanisms that involve the same structures can be utilized for all forms of behavior. What, therefore, constitutes abnormal behavior? When people speak of normality, they actually have averages in mind. They do not generally, however, regard every deviation from the average as abnormality. For some forms of behavior, public opinion determines the limits of normality.

After more careful thinking, a borderline zone of doubtful normality is recognized. Then the mores alter, and what was abnormal is deemed normal. The fact is that though variation is the basis of growth, the person who varies from the mass is abnormal, but he is the hope of humanity if his energy can be collectively capitalized.

During the time of war he who advocates peace is abnormal in his behavior, but his behavior may represent normality of intelligence, judgment and ideals. It is normal for children to play, but if they wish to play all the time, it may be regarded as abnormal. Sleep is normal behavior, but to sleep or want to sleep for unusual periods of time is deemed abnormal. For a boy to be unwilling to fight in self-defense is abnormal, because society puts a certain value on a behavior type that is not too submissive.

Society is responsible for what constitutes norms of normality. If one breaks a leg and limps, there can be no question about the physical deviation. If one has sibilant râles, all his asthmatic behavior is an abnormal condition. A girl 5 feet 11 inches (180.3 cm.) tall has height above the average, and many of her reactions to this may be regarded as abnormal socially, when they are normal and natural reactions for one of her height.

What is normal in one place may not be regarded as normal in another. The standards of height of Japan would be distinctly abnormal for Norway. Menstrual behavior among the Eskimos and the East

Indians is different. The behavior of primitive people, such as the Maori and the Papuans, would be regarded as abnormal among the Anglo-Saxons. Infanticide and wife beating, trial marriages, stealing and blood feuds are today deemed abnormal behavior, though all of them have had social sanction and were held to be normal and socially desirable at different times and in different places.

Behavior as conditioned by intelligence has wide standards of variation. Intelligence in turn is conditioned if it is not determined to some extent by physical structure and function. A Kalmuck may have normal intelligence for his level of existence just as do the Laplanders, the Patagonians, the Aleutian Islanders and the natives of Borneo. These intellectual levels, however, are not identical with those deemed normal in Germany, Turkey, Japan or the United States. The behavior resulting from these different degrees of intellectual capacity and development creates numerous difficulties when any one of these groups is transported beyond the confines of his particular mores. It is evident that among primitive races and highly developed groups the physical differences are less pronounced than are the intellectual variations. The gross physical reactions, therefore, do not explain individual or group behavior.

INTERPRETATION OF BEHAVIOR

Behavior must be viewed socially and must be interpreted in terms of time, place and circumstance. Behavior per se is neither good nor bad. Scarcely a vice can be mentioned, which, under certain conditions, has not been deemed a virtue. "Goodness" may be pathologic and abnormal behavior. Few forms of abnormality are without a counterpart in normality. What is abnormal here may be normal elsewhere.

Recognizing, then, that the use of the term abnormal behavior raises many questions, let me define abnormal behavior as a variation from accepted standards of behavior. The accepted standards usually represent the forms which are socially acceptable to a group of people as a result of their traditions and experience. In some instances, a pattern of behavior represents the actual practice of 100 per cent of a community. Under other conditions, the standards may represent the ideals but not the practices of the community. Abnormal behavior would be that which runs counter to the views, beliefs, habits and customs of at least 60 or 75 per cent of the people.

Abnormality may be differentiated into certain spheres. If one regards conscious, volitional man as animated machinery, abnormalities in behavior may be found in any one of four general realms. Physical deviations deal with the mechanics of the organism, with the internal frictions reflected by emotional reactions. Intellectual processes would represent the operator of the machine, while the social variations of

behavior would represent the type of work accomplished by the machine in and on the environment. The machine may and does function abnormally in each and all of these directions. If, for the moment, one considers this reacting vital machinery in terms of its conscious and subconscious life, one may properly ask what are the physical problems that affect it.

EFFECT OF PHYSICAL DISABILITY ON BEHAVIOR

What does the word physical mean? I have already referred to the fact that the term physical can be defined only in terms of structure and function. Functional restorations today are deemed of more importance than structural restorations, though one in a sense conditions the other. Normal function grows out of and preserves normal structure, and normal structure grows out of and tends to preserve normal function. Neither of these facts is absolutely true, because of the numerous interrelations which exist among all parts of the human mechanism. Structural variation may be exceedingly temporary, as in a hair cut; functional disability may be equally brief in any hysterical blindness; both structure and function may be transiently modified, as in an urticarial wheal. The interrelation between structure and function is exceedingly close, and this becomes all the more noticeable in the psychologic field that involves both conscious and unconscious behavior. The physical phases of behavior constitute merely a facet of life. Behavior in man is the expression of a psychobiologic unity. An idea may paralyze movement, and a movement may paralyze ideas.

A child has lost a thumb. This would appear to be a true physical disability. The thumb of the right hand is gone—a simple physical fact. But is it only the thumb that has gone? Something has been subtracted from the personality of the child. He is now different. By the law of averages, he is an abnormal physical being. With the loss of his thumb, there was a loss of equality which will require compensation in many ways. He will not be able to throw a ball, to handle certain tools, or to indulge in forms of manipulations which are permitted to others of his age and sex. He may derive a sense of incapacity or inferiority which may lead to patterns of action distinctly antisocial. His behavior has been physicially conditioned by the loss of the thumb, which, in turn, has involved numerous psychic adjustments.

John has a cleft palate, but the failure of union of the bones resulted in a cleft in his being. The operative procedures with resultant pain and distress, his consciousness of an unpleasant voice, and his painful attitude toward those who mimic it had brought out an aggressively defensive, pugnacious, resentful, sensitive and unhappy personality. Literature has utilized the nose of Cyrano de Bergerac and the deformities of Richard III. to point out the effect of physical deviations on behavior and character. Rachitic deformities, alopecia areata and poly-

dactyly are not confined to bones and skin. They are obvious conditions that work on the entire personality. Cryptorchidism, epispadias and hypospadias, even though they may be regarded as simple physical developmental anomalies, are fraught with diverse effects on behavior. The possibility of surgical treatment of these conditions involves a consideration of much more than the mere retention of the testes in the scrotum, or the functional restoration of the urethra.

The total effect of a physical disability is rarely predictable. The nature of the physical factor does not indicate its effect on behavior any more than one may be certain concerning the metastases of some forms of tumor. Tuberculosis and syphilis, for example, present a variety of places of involvement. Even the site of pathologic processes does not suffice to indicate what behavior reactions will necessarily follow. A variety of disease processes may produce the same psychologic results, and the same disease processes of the same organ may produce different psychologic results. The structural similarities of persons do not guarantee functional identities, except in terms of a few viscera. The process of digestion, for example, proceeeds identically, even though the visceral topography is not uniform. A slight variation in gastric acidity may cause profound differences of behavior attitudes and emotions in different persons. A seemingly mild physical injury may cause profound disturbances in the sphere of physical, intellectual, emotional and social activities. Traumatic neuroses afford abundant illustrations. The physical problems of behavior are as extensive and as complicated as the physical and psychologic nature of man.

Some specific physical disabilities create problems that are exhibited in all the realms of human activity. Encephalitis or a fractured skull, for example, may affect and seriously alter behavior in its four major realms. A variety of physical limitations including convulsions may result, accompanied by marked mental deterioration, emotional hyperactivity and antisocial conduct such as pyromania, theft, cruelty, lust and crime. Cretinism lowers the general level of activity and behavior in all of the fields. Nevertheless, one must be careful not to assume that a particular form of abnormal behavior is directly attributable to the obvious physical condition, because similar forms of misbehavior may be present on a psychogenetic basis. Lying and stealing, for example, must not be regarded as due to encephalitis, unless there has been a profound alteration of personality in a child previously truthful and honest. Concomitant disease and underlying disease must be differentiated in interpreting behavior.

Some physical disabilities mainly affect the purely physical expression of activity. Flatfoot, for example, or the mild scoliosis which is so often seen at adolescence usually does not have marked effects on behavior, although both may increase fatigability and thus heighten irritability.

An ankylosis of the jaw or elbow represents a more profound type of physical disability, but the slight limitations thus arising may not bring about noteworthy changes in the emotional and social sphere, while marked limitations may bring about distortions of behavior as well as of contour.

A second type of physical difficulty may spend its main effects on the mental element in behavior. Birth traumas, for example, with intracerebral hemorrhages, bring about a type of behavior that is partially conditioned by the actual paralyses, but more particularly by the interference with mental function. The intellectual development of the child with birth traumas is more important than the amount of spasticity that remains. The acquired limitations of cerebral function markedly influence the general welfare. It is not without some significance that the cripple has a prominent place among vagrants. Defective vision, as a purely physical fact, handicaps the development of inherent brain power. The limitations of intellectual activity, together with the effects of the actual physical strain in visual attention, tend to make visual errors responsible for widely different types of abnormal behavior. For example, the same degree of myopia in different children may produce fidgetiness, irritability and disobedience, or sleepiness, weepiness and submissiveness.

Another group of physical conditions, and I am merely hinting at some of the physical problems of abnormal behavior, may create the major problems in the realm of the emotions. For example, a variety of orthopedic conditions that handicap free movement and limit the satisfaction of psychic needs and desires warp the emotions. A short or shriveled leg, an ankylosed joint, a crutch or a brace are powerful levers in emotional life. Defective hearing profoundly affects emotional life, as a result of which there is a natural tendency to shrink from social contacts. Persons who are hard of hearing present forms of behavior which require considerable adjustment, largely because the instruments for correcting this deficiency are not comparable with those available for defects in vision. A large nevus on a girl's face has little physical effect, but it may sadly demoralize her emotional development. An ichthyosis or ezcema may be more devastating in its emotional reactions than the knowledge of a gonorrheal infection or a syphilitic involvement, both of which at times cause outstanding emotional distress with gross abnormalities of behavior, even to the extent of precipitating serious neuroses or psychoses.

Still another type of physical disability conduces to problems of behavior primarily in the realm of social activity. When, for example, dwarfism exists, structure and function may be normal, but the characteristics of dwarfism in themselves constitute physical abnormality, and, as such, a complete adjustment in social relationships is funda-

mentally involved. The restriction of vocations, conspicuousness in groups, the limitations of physical prowess, all combine to bring about types of behavior which are abnormal only to the extent that society has made conformity a virtue.

There is a marked tendency to explain all types of behavior as due to endocrine function or dysfunction. The whole subject of endocrinology is still in a state of clinical chaos. Comparatively few of the behavior difficulties of children are proved to be primarily due to disorder of the ductless glands. In some instances, glandular dysfunction and abnormality of behavior may coexist; they may be dependent on an identical underlying cause, or they may interact on each other. To attempt to relieve all the forms of abnormal behavior by glandular therapy is unwise and irrational. The coexistence of physical phenomena does not establish causal relationships.

Spina bifida, with its consequent lack of control over urination and defecation, obviously hampers normal development of the finest forms of social behavior.

COMMENT

I have sought to indicate that the physical problems of abnormal behavior are not isolated in human welfare. I have not dwelt on the effects of ideation or psychic function on physical behavior. It was not germane to discuss the development of the neuroses and psychoses in terms of physical causation, nor, indeed, to present the principles involved in psychogenetic functioning. I have avoided all reference to the theories of Freud, Adler and Jung, and the constitutional theories of Kretschmer, because it seemed to me that the actual place of physical problems in abnormal behavior merited more adequate attention. I have purposely avoided discussion of organic lesions of the cerebrospinal nervous system as being too obviously a physical factor in behavior. I have sought merely to stress the importance of recognizing the interrelation of the structure and function of the physical system, always considered, however, in relation to the totality of living. While behavior, in a sense, represents action and in another sense represents inaction, inhibition is active inactivity. What man does and what he does not do are not dependent entirely on his structure, nor is the function or lack of function of any structure limited to that particular physical unit. Behavior is the sum total of reactions. Hence, the physical variations and deviations of man as a reacting, adapting mechanism require interpretation in the light of his intellectual, emotional and social growth. Physical disease may be generic, but behavior is individual, though its measure lies in socially developed standards. Man's activities may be diversified in terms of structural and functional unit systems, but he lives and functions as a whole, a psychobiologic entity.

Pediatric Biographies

SORANUS OF EPHESUS

JOHN RUHRÄH, M.D.
BALTIMORE

The ancients were not unmindful of infants and young children. In many of the early texts that have been handed down in more or less mutilated form, there are references to the hygiene of early life and accounts of diseases peculiar to the young. In the medical writings of the Egyptians, as in the Eber papyrus, in the Hindu books, in the Syriac "Book of Medicine," in the writings of Aetius, Paulus Aginetas and others, there are extracts of pediatric interest. Hippocrates did not neglect this subject, and Troïtzki [1] has made an interesting study of the passages relating to children.

But after all these and other writings have been considered, there is one early pediatric text that stands out in its clearness and accuracy. This is the writing of Soranus relating to the hygiene of infancy which forms a part of his Περὶ γυναικείων. Perhaps the most useful text for the modern student is the German translation by Lüneburg,[2] which is to be found in the first volume of the Bibliothek Medicinischer Klassiker edited by J. C. Huber. There are other texts, and much has been written about them, but a discussion of these is of linguistic rather than of medical interest.

Who was Soranus? Little is known about him except that he came from Ephesus and that he imbibed Greek culture there and at Alexandria. He settled at Rome between 110 and 130 A.D., and then he disappeared. It is to be hoped that he settled down to a peaceful old age on a sufficient competence.

Soranus is generally associated with that sect of medical practitioners called the methodists, but he was an independent observer and thinker and much too broadminded to be tied down by the tenets of a medical creed. He was a prolific writer and is credited with more than thirty volumes. Unfortunately, only some of his gynecologic and obstetric articles have been preserved. Nothing of any moment was added to what Soranus knew about obstetrics until about fifteen hundred years later.

His pediatric writings made a deep impression on medicine, and what he wrote was copied and rewritten by subsequent writers. A

1. Troïtzki: Hippocrates als Kinderarzt, Arch. f. Kinderh. **29**:223, 1900.

2. Soranus: Textbook, trans. by Lüneburg; Bibliothek Medicinischer Klassiker, München, J. F. Lehmann, 1894, vol. 1, chapters 26 to 48.

Sorani Ephesii

de

Arte obstetricia morbisque mulierum

quae supersunt.

Ex apographo

Friderici Reinholdi Dietz,
Med. et Chir. Dr., Medicinae in Academia Prussorum Albertina
Professor. Ordinar. etc.

nuper fato perfuncti
primum edita.

Regimontii Prussorum
in Commissis apud Graefium et Unzerum.
MDCCCXXXVIII.

Title page of Dietz' edition of Soranus.

careful examination of the early texts reveals much that was credited to Soranus, but who knows whether it originated with him or was taken by him from some earlier source?

Troïtzki [3] has analyzed each chapter and has made a comparison of what Soranus stated with what was currently believed at the end of the nineteenth century and with the progress that had been made in the intervening time. There are 127 pronouncements, and of these nearly one-half, sixty-two, are still regarded as sound. How many of the present day books will stand such a test after a similar lapse of time?

One short extract will give some idea of his way of treating his subject. Of course, no account of Soranus, however short, is complete without something about his finger-nail test for milk; the original is given below, but it may not be amiss to quote a more modern paraphrase:

> Soranus, he of ancient Rome,
> He had a simple trick,
> To see if milk was fit for sale,
> He merely dropped it on his nail
> To see if it would stick;
> Yet spite of this the babies grew
> As any school boy'll tell to you.

I. NUTRITION OF THE NEW-BORN INFANT

After the infant has been swaddled and cradled, it should rest and receive no food at least for the first two days, for the child is apt to be in continual motion during all this time and its body is still amply provided with nourishment derived from the mother, which it must first have time to digest before it is ready for new food. The case is altered should it develop a premature appetite, the signs of which I will explain later. After this pause, something may be prepared for the child to lick up, but never butter, which is difficult to digest and injurious to the stomach. . . . For this purpose, slightly boiled honey is much more to be recommended for everything raw is sharp and produces flatulence; but things cooked too long are constipating in the highest degree, and what is cooked for a moderate length of time cleanses the stomach and the intestines. The mouth of the nursling is, therefore, to be softly stroked with the finger after which luke-warm honey and water is dribbled into it. In this way, the rich, thick material is diluted, the appetite is increased, since the child will recollect the pleasant taste, the oesophagus is opened, the digestion of food proceeds easily after the canal has been cleansed and thus the whole constitution is nourished. (After the nursling has been cared for in this manner for these two days, it can be given milk on the next and following day at the breast of a good wet nurse.) During the first twenty days the mother's milk is, as a rule, unfit for consumption by the child, being thick, cheesy and difficult to digest.

II. THE FINGERNAIL AND WATER TESTS FOR MILK

Whether the milk will coagulate properly is determined by the fact that if we put a drop on the fingernail or on a laurel leaf or other smooth surface it slowly

3. Troïtzki: Arch. f. Kinderh. **17**:95, 1894.

spreads and when shaken, retains the drop-form; for if it flows at once in all directions, it is watery, but if it coheres like honey and does not change its drop-form, it is too thick. We may also make the test by adding twice as much water to a given quantity of milk; the solution will then take place only after a considerable time and the white color is retained to the last. If solution takes place at once the milk is watery and it can be used still less if it forms a fibrous coagulum like that of serum. In such a condition it is indigestible. But if, after sometime, it does not diffuse and sinks in such a manner, that it remains at the bottom when water is poured on it, it is cheesy, thick and soluble with difficulty.

News and Comment

RACE BETTERMENT CONFERENCE

The third race betterment conference was held at Battle Creek, Mich., from Jan. 2 to Jan. 6, 1928. The object of the conference was to introduce the work of the scientist directly, not only to the layman, but to the physician and to the educator with whom the layman comes more directly in contact in everyday living. Many persons prominent in both science and education were scheduled as speakers.

MID-WEST CONFERENCE ON CHILD STUDY AND PARENT EDUCATION

The Mid-West Conference on Child Study and Parent Education under the auspices of the Chicago Association for Child Study and Parent Education will be held in the Palmer House, Chicago, February 16, 17, 18. The main theme of the conference, "Character Development," will be discussed by eminent speakers and also by authorities conducting round tables. The following tentative program includes lectures by well-known authorities on Child Psychology:

Thursday, February 16.

Morning: 1. Various Phases of Character Development. MARK A. MAY.

2. The Science of Character Building (keynote speech). E. D. STARBUCK, University of Iowa.

12:15-2:30: Round Tables (approximately five).

Leisure Time Activities and Their Influence on Character Building.

Education for Courage.

Afternoon: Two sessions. Craftsmanship and Character.
Speakers to be announced.

Friday, February 17: Emotional Health of the Child.

Morning: 1. The Parent and the Child's Emotional Social Adjustment. FLORENCE MATEER, Children's Aid Society, Columbus, Ohio.

2. Relation of Success and Failure to the Emotional Life of the Child. BERNARD GLUECK.

12:15-2:30: Round Tables on Religious Education.

Afternoon: 1. Constructive Aspects of Emotional Conflict. WILLIAM HEALY.

Evening: Dinner Meeting. Title to be announced.

1. HAROLD RUGG, Columbia Teacher's College.

2. Speaker to be announced.

Saturday, February 18: Special Emphasis on Teacher's Influence as Well as Parent's.

Morning: 1. Discipline and Character. WILLIAM KILPATRICK.

2. Ideals and Character. Speaker to be announced.

Further information will be given by the office of the Chicago Association for Child Study and Parent Education, 537 South Dearborn St., Chicago; Telephone, Wabash 7354.

DR. ABT MADE OFFICER OF LEGION OF HONOR

On the afternoon of Friday, November 4, at the Church Library, Northwestern University Medical School, Doctors J. M. Le Mee and G. Illingworth Helie, acting as representatives of the French government, conferred on Dr. I. A. Abt, Professor of Pediatrics, Northwestern University Medical School, the decoration of an officer of the Legion of Honor for his attainments in pediatrics.

CLINIC FOR THE STUDY OF ASTHMA, ECZEMA AND HAY-FEVER

The New York University has announced the opening of a clinic for the study of asthma, eczema and hay-fever in children at the New Sydenham Hospital, 123rd Street and Manhattan Avenue. The clinic will be under the direction of Dr. Bret Ratner.

ELECTION OF DR. PIRQUET

Friends of Dr. Clemens Pirquet will be interested to know that he has been elected president for the ensuing year of the International Union for Child Welfare, a bureau of the League of Nations. Professor Pirquet is at present director of the children's clinic at the University of Vienna and in his new position will succeed the Duke of Atholl.

PAN-AMERICAN CHILDREN'S CONGRESS

The fifth Pan-American Children's Congress will be held in Havana, Feb. 13 to 20, 1928.

CORRECTION

In the article by Dr. William L. Rost, "Generalized Edema of Undetermined Etiology: Report of an Unusual Case" (Am. J. Dis. Child. **34**:773, 1927), the word "normoblast" in the third column of table 1, p. 775, should read "normal."

Abstracts from Current Literature

Anatomy and Physiology

CONTRIBUTION TO THE ANATOMY AND FUNCTIONS OF THE HYPOPHYSIS. II. THE NERVOUS REGULATION SYSTEM OF THE POSTERIOR LOBE OF THE HYPOPHYSIS. THE NUCLEUS SUPRA-OPTICUS AND ITS FIBER SYSTEMS. R. GREVING, Ztschr. f. d. ges. Neurol. u. Psychiat. **104**:466 (Sept.) 1926.

In 1922, Greving described the fiber systems of certain cell groups, among which was the nucleus supra-opticus. Besides the tractus supra-opticus superior, which formed a part of the lower thalamus, he described the tractus supra-opticus inferior. According to his description then, this tract passed around the medial side of the optic tract to the tuber cinereum, where it was lost between the cells of the substantia grisea centralis and the cells of the tuber cinereum.

In 1925, he demonstrated a fiber bundle which ran from the vicinity of the nucleus supra-opticus through the tuber cinereum into the infundibulum and posterior lobe of the hypophysis. This tract he designated as the tractus supra-opticus-hypophyseus; it is identical with the tract described, in 1922, as the tractus supra-opticus inferior. J. L. Pines, in 1925, confirmed these observations. Greving attempts in this present contribution to determine more about this tract, and to find out if other fiber systems are connected with the nucleus supra-opticus. He concludes that: (1) The nucleus supra-opticus surrounds the optic tract at the foot of the midbrain, and that it consists of cell groups, a pars dorsolateralis, dorsomedialis and ventromedialis. These cell groups, however, are connected with one another and form a single cell area which extends down as far as the tuber cinereum. In this way one can understand why Lewy obtained pathologic cell changes in the tuber cinereum after a lesion of the posterior pituitary lobe. (2) From the large cell territory of the nucleus supra-opticus there arises a large fiber bundle, the tractus supra-opticus-hypophyseus, which passes through the tuber cinereum to the infundibulum to enter the posterior lobe of the pituitary. "The assertion of Lewy, therefore, of a connection between the posterior lobe of the hypophysis and the nucleus supra-opticus is demonstrated in serial sections in man anatomically." (3) At the cell area of the nucleus supra-opticus certain fibers are acquired which come from the gray matter of the third ventricle. These arise from the nucleus paraventricularis from the vicinity of the chiasm almost to the commissura intermedia. This tract is designated as the tractus paraventricularis-cinereus. It ends in the vicinity of the nucleus supra-opticus. (4) The nervous regulation of the posterior lobe of the hypophysis arises, therefore, from the nucleus paraventricularis, tractus paraventricularis-cinereus, nucleus supra-opticus, and the tractus supra-opticus-hypophyseus.

ALPERS, Philadelphia.

Pathology, Bacteriology and Biochemistry

A STUDY OF THE COAGULATION OF THE BLOOD; THE CHEMICAL REACTIONS UNDERLYING THE PROCESS. A. B. WADSWORTH, F. MALTANER and E. MALTANER, Am. J. Physiol. **80**:502, 1927.

The writers have found that in the coagulation of blood, certain definite chemical reactions take place. First there is a reaction of ionized calcium salts with an active lipoid substance to produce a calcium-lipoid complex and free acid. In the presence of the latter, the proteins of the plasma react with lipoid to produce an insoluble lipoid-protein complex-fibrin. The rôle of the calcium is apparently that of a catalyzer, since the amount required is

small, and since it is found in almost undiminished amount in the serum after termination of the clotting process. They suggest that three distinct phases are involved in the process: first, the combination of lipoid with calcium salts, with liberation of the free acid; second, a reaction between the free acid and fibrinogen to produce acid fibrinogen; and, third, a reaction between the acid fibrinogen and the calcium-lipoid complex, with liberation of the calcium in combination with the acid.

H. E. Eggers.

The Rôle of the Thyroid and Parathyroid Glands in the Chemical Differentiation of Bone During Growth. F. S. Hammet, J. Biol. Chem. **72**:505, 527, 1927.

Thyroid and parathyroid deficiencies produce distinct types of distortion of the chemical differentiation of bone, with respect to ash, organic matter and water, during growth. Using the percentage of ash as the index of ossification, both types of glandular deficiency produce a lesser degree of ossification than that found in bones of animals of the same age that have not been operated on. Most of this is determined by the retardation of growth of the bone. On the basis of the stage of development as expressed by weight of the bone, it is only in the case of parathyroid deficiency in the female that ossification is less in degree than normal. Parathyroid deficiency initiated during the pubertal adjustment in the male, conditions a decisive shift in chemical differentiation. This is traceable to the increased sex-specific sensitivity of the organism as a whole.

A distortion of chemical differentiation of bone with respect to calcium, magnesium and phosphorus follows thyroparathyroidectomy and parathyroidectomy in albino rats. The nature of the distortion is the same in both groups, but less in degree in the former. It produces a bone of lower calcium and phosphorus, and a higher magnesium percentage than that normal for the age. The ash has a higher magnesium and phosphorus, and a lower calcium percentage. The shifts in the thyroparathyroidectomized groups are attributable to the parathyroid deficiency. From the point of view of the growth of the bone, however, the specific influence of parathyroid deficiency on calcium increment is not exhibited when the thyroid is likewise lacking. On the basis of stage of development as expressed by the weight of the bone the deviations from the expected percentage values are in general the same in kind as those for ash and are similarly interpretable. Parathyroid deficiency over long periods does not result in decalcification of the bones. A particularly important phenomenon is the relatively greater disturbance of the magnesium fraction.

Author's Summary.

The Influence of Purulent Infection on the Development of Experimental Scurvy. Henry L. Jaffe, J. Infect. Dis. **40**:502, 1927.

Six of eleven infected guinea-pigs on a scurvy diet developed severe purulent infections and did not develop scurvy within from nineteen to forty days. Uninfected guinea-pigs on the same diet developed scurvy in from thirteen to eighteen days. The injection of *Staphylococcus albus* into animals with early signs of scurvy caused a rapid fatal termination. The course of scurvy was not influenced by the injection of standard staphylococcus vaccine. These studies suggest the possibility that antiscorbutic substances may be developed by bacterial growth in the tissues or as a result of the action of absorbed toxins on the internal organs.

Author's Summary.

Phenol and Tricresol Effects in Toxin and Antitoxin Solutions. C. G. Pope, J. Path. & Bact. **30**:301, 1927.

A separation of phenol can be shown to occur when a 0.5 per cent phenol solution is frozen. The extent of the separation of phenol depends on the protein and salt content of the toxin or antitoxin solutions, the freezing tempera-

ture and the duration of freezing. When these factors permit, a critical amount of phenol may separate from a toxin-antitoxin mixture, causing a greater relative destruction of antitoxin than of toxin and rendering the mixture toxic. A separation of phenol can be shown to occur at the air liquid interface when a 0.5 per cent solution of phenol in water or toxin is shaken. This separation of phenol causes marked destruction of the specific substances when dilute solutions of toxin or antitoxin containing these antiseptics are shaken in a partially filled bottle. The use of phenol or tricresol in material for the Schick test or for the Schultz-Charlton test is therefore inadvisable.

AUTHOR'S SUMMARY.

DIPHTHERIA TOXINS PRODUCING A DIPHTHEROID BACILLUS. P. HAUDUROY, Compt. rend. Acad. d. sc. **184**:409, 1927.

There is a filtrable form of the diphtheria bacillus which may be identical with the toxic principle found in the sterile filtrates of broth cultures of this organism. The filtrable form may be isolated and grown by inoculation of the toxic filtrate into an especial medium found successful for the cultivation of other filtrable forms. It reverts by fusion into a nontoxicogenic diphtheroid modification.

ARTHUR LOCKE.

PROBLEMS IN DIPHTHERIA. I. THE BIOLOGY OF THE CORYNEBACTERIA AND THE QUESTIONS OF VARIABILITY. M. GUNDEL, Centralbl. f. Bakteriol. **101**:337, 1927.

Gundel claims that in the animal body diphtheria bacteria may become transformed into pseudodiphtheria bacilli and that in vitro these may be changed back into true diphtheria bacilli. In vitro, a pathogenic diphtheritic strain may be transformed to a nonpathogenic strain and vice versa, and a pseudodiphtheritic strain into a nonpathogenic diphtheritic strain. He feels that the virulence test of diphtheritic strains in guinea-pigs is no criterion as to their virulence for human.

PAUL R. CANNON.

SIGNIFICANCE OF EXPERIMENTAL COOLING OF THE BODY WITH REFERENCE TO COLDS. LUDWIG FLEISCHER, Ztschr. f. Hyg. u. Infectionskrankh. **107**:2, 1927. BACHMANN and LUDWIG FLEISCHER, ibid., p. 28.

The author reports on a large series of experiments on himself concerning the relations between skin temperature and warmth of the surrounding atmosphere. At the same time he carefully observed his general condition during the course of the experiments. These observations were made while he was sitting quietly, either in his ordinary clothes, in light clothes or without clothes. He measured the temperature of the skin over the extensor surface of the basal phalanx of the left middle finger and of that over the third intercostal space close to the sternum. A third measurement which he considers to be particularly important is that of the temperature between fingers because it gives some indication of the temperature of the deeper tissues. As long as the finger temperature did not decrease below 30 C. he did not notice any general effects except perhaps a slight feeling of coolness. In ordinary clothes the surrounding temperature in the absence of any drafts was indifferent between 18.5 and 21 C. In the afternoon both limits were lower. When the finger temperature rose above 33 C. he felt too warm. When the finger temperature dropped to 25 C. his hands felt cold. When it fell below 21 C. he began to feel cold generally. If at this point the experiment was interrupted the finger temperature under favorable conditions rose to 30 C. and his feeling of well being was completely restored. If the finger temperature had gone down below 20 C., general disturbances continued for the rest of the day such as: backache, coldness of the back, drowsiness and marked depression. At a finger tempera-

ture of 8.5 C., cramps developed in the muscles of the hands. In ordinary clothes and with an indifferent temperature in the surroundings the temperature between fingers was from 1 to 1.5 C. higher than the finger temperature. When the finger temperature decreased the difference between the two temperatures decreased until they were almost equal. The skin reacted in the same manner during a period of six months. It did not become more resistant to cooling but the sensitiveness to cold was somewhat reduced. The influence of hot drinks and of alcohol on the experimental results was interesting. From 15 to 25 cc. of alcohol in the form of alcoholic liquor or diluted absolute alcohol of approximately room temperature produced a strong subjective sense of warmth, raised the finger temperature but not that of the skin over the chest, whereas hot alcoholic and nonalcoholic drinks of a temperature of about 60 C. in quantities of about 300 cc. had exactly the same effect on the temperature of the fingers and also raised the temperature of the skin of the trunk. Hot nonalcoholic drinks were more valuable to correct the ill effects produced by cooling; they also prevented them to some extent when taken before the experiment was started. Hot water had the same effect as coffee or tea.

The observations which Fleischer had made on himself in regard to the relation of the temperature of the skin of the fingers to the general effects of exposure to cold were confirmed on other persons, also that hot drinks counteracted the effects of exposure, while the taking of cold alcoholic beverages was not equally efficacious. Neither one of the authors contracted cold during these experiments, although both were susceptible to cold as shown by the fact that during the period of experimentation they contracted colds in other ways. Observations on other persons, however, seemed to indicate that an acute attack of cold may follow a sudden reduction of body temperature by a cold wind or rain. Colds contracted in this way apparently are not infectious.

W. Ophüls.

Proliferative Reactions of the Thymus. V. Wituschinski, Virchows Arch. f. path. Anat. **262**:595, 1926.

The reactions set up in the thymus by the presence of aseptic foreign bodies were studied in young cats and rabbits. The immediate change which resulted from the introduction of the foreign body, a spicule of celloidin, was necrobiosis of the lymphoid cells. Within seven hours, the cells of the epithelial reticulum became enlarged and phagocytized the necrotic lymphocytes. This was followed by proliferation of the epithelial reticulum, of the periadventitial histiocytes and, to a lesser degree, of the endothelium of the capillaries. The young epithelial reticulum cells differentiated into fibroblasts, as did also the histiocytes, and formed a collagenous capsule about the foreign body. Histiocytes and perhaps also young epithelial reticulum cells formed myelocytes and pseudo-eosinophilous granulocytes. Plasma cells were formed from the adventitial histiocytes.

O. T. Schultz.

A Comparative Study of Chicken and Pigeon Pox. T. Saito, Ztschr. f. Immunitätsforsch. u. exper. Therap. **48**:451, 1926.

Saito describes a comparative study of two strains of virus of chickenpox and two of pigeonpox. Chickenpox virus, when it "takes" in the pigeon, causes a reddening and swelling of the skin with small nodules. The virus, however, can be transmitted to other pigeons and causes a spreading eruption. This passage attenuates it for the chicken. When pigeonpox virus is applied to the skin of a chicken, the reaction is slight. Further passage makes the reaction even weaker, but the virulence for the pigeon remains unchanged. There is a clear-cut reciprocal immunity, however, each of the species being made immune to both viruses by immunization with one.

Paul R. Cannon.

Nutrition

INCIDENCE OF MASTITIS AND THE INFECTING ORGANISMS IN FOUR DAIRY HERDS. JOHN G. HARDENBERGH and CARL F. SCHLOTTHAUER, J. Infect. Dis. **40**:667, 1927.

The results of the study of sixty-eight cases of mastitis indicate the prevalence of streptococci as infecting agents in the disease, although other organisms, particularly staphylococci and members of the colon-typhoid group, may show occasional periods of predominance. The veterinarian seldom sees cases of bovine mastitis that are not complicated by infection. It is believed that the incidence of the more severe cases of mastitis can be materially reduced in all herds by adherence to a few simple rules of dairy hygiene, such as the isolation of animals with mastitis, the avoidance of disseminating the infection from infected cases, the prevention of dietary disorders and precautions against traumatism.

EXCRETION OF ANTISEPTIC DYES THROUGH THE MAMMARY GLAND. VICTOR BURKE and E. A. RODIER, J. Infect. Dis. **40**:673, 1927.

Neutral acriflavine was excreted through the mammary gland of the cow following the intravenous injection of from 7 to 11 mg. per kilogram of body weight. The milk was well colored. The injection of 11 mg. per kilogram caused a severe reaction and a temporary cessation of the milk flow. Although the experiments failed to demonstrate an increase in the bacteriostatic action of the milk following the intravenous injection of neutral acriflavine, there is reason for believing that injection of the dye may affect the organism in the udder.

The intravenous injection of gentian violet caused more severe reactions than acriflavine, and mercurochrome-220 soluble caused still more severe and prolonged symptoms. Apparently, these dyes were not excreted by the mammary gland.

TYPES OF HEMOLYTIC STREPTOCOCCI IN CERTIFIED MILK. WILLIAM D. FROST, MILDRED GUMM and ROBERT C. THOMAS, J. Infect. Dis. **40**:698, 1927.

The milk from about 1,000 cows on five different certified farms has been plated on blood agar about once a month throughout the period of one and one-half years.

The samples were from groups of ten cows. Hemolytic streptococci of the true beta type appeared in 28.6 per cent of the samples. The number of such streptococci in the samples was usually small: in 65 per cent, less than 1,000 per cubic centimeter, and in 94 per cent, less than 10,000 per cubic centimeter.

Classification of 394 cultures of the true beta type, by means of seven different tests, placed them in six different species. Five of these, the mastitidis, infrequens, subacidus, hemolyticus I and epidemicus have been described before. One new species is described, *Streptococcus asalignus.*

All of the identified streptococci found in certified milk are of the bovine type with the exception of four cultures which proved to be *Streptococcus epidemicus* Davis.

The method of procedure used in this work for examining milk and identifying streptococci promises to be of distinct value in the production of milk of the highest quality.

ARCH. PATH.

THE ETIOLOGY OF RICKETS. R. SIDEN, Practitioner **119**:45 (July) 1927.

It cannot yet be said that the cause of rickets is definitely known. It is the author's opinion, due to the specificity of rickets, that a primary cause will be found and that the many factors now mentioned will be found to be simply predisposing causes.

The whole trend of medical opinion today is toward the view that the deficiency of solar ultraviolet rays is the factor of paramount importance and that dietetic and other factors are of minor importance.

The geographic distribution of rickets is in harmony with this hypothesis. The way in which light acts on the body is still unknown, but it is probable that cholesterol or a similar substance plays an important part in the process.

The reaction that occurs when an infant's skin is exposed to ultraviolet rays is a photochemical reaction that takes place in the skin itself.

It seems certain that the deficiency of the antirachitic element in the body is the cause of rickets. The vitamin can be supplied in the diet, or the body itself, if it is exposed to ultraviolet radiation, could synthesize the vitamin.

The solar ultraviolet rays penetrating the superficial layers of the epidermis act on the vitamin precursor and by means of a photochemical reaction convert it into the antirachitic vitamin. This vitamin is absorbed by the blood and, acting like a hormone, controls the metabolism of the body, particularly the deposition of calcium salts in the bones. Although this hypothesis cannot be regarded as more than a mere surmise until further evidence is available, yet it seems to be consistent with clinical evidence and is not negatived by experimental observations.

GELSTON, San Francisco.

NUTRITION, GROWTH AND FEEDING OF INFANTS; PATHOGENESIS OF "DYSTROPHY" FROM COW'S MILK. DANTE PACCHIONI and ANTONIO ROSSELLO, Riv. di clin. pediat. **25**:529, 1927.

Nutrition appears to be a unique complex chemical process guided by the neuro-endocrine system; it is influenced not only by digestion and absorption but also by all the intrinsic (constitutional) and extrinsic (environment and needs of nourishment) factors which affect the metabolism of the tissues.

Dystrophy resulting from the ingestion of cow's milk is caused by an upset of the whole nutrition and is a consequence of the strong tendency of the infant to grow. Cow's milk in these cases fails to furnish either qualitatively or quantitatively sufficient nourishment to meet the needs for growth.

HIGGINS, Boston.

NUTRITION OF THE CHILD IN THE FIRST TWO YEARS OF LIFE, WITHIN AND WITHOUT CLOSED INSTITUTIONS. H. KLEINSCHMIDT, Gesundh. f. d. Kindesalter **2**:330 (June) 1927.

Kleinschmidt pointed out that there are three artificial forms of infant feeding at present in use: first, the ordinary milk dilution formula; second, the concentrated food mixture, such as the butter flour mixture and the dubo; third, the lactic acid milk modifications. He suggested that success with the first type depends on excellent nursing and an absence of infections, and believes that in general, better results are obtained outside of institutions than within them. After being put on these mixtures in instituions, children often show attacks of dyspepsia. Kleinschmidt suggested that these may not always be due to parenteral infections but may be a reaction to an unfavorable change in environment. He believed that the more closely the institution copies the previous habits of the child, the less often one sees such results. These changes are less frequently seen in the case of concentrated food mixtures. One also sees better increase in weight. On the other hand, it is likely that these foods give the infant too many calories at home, since here it is likely that the child has a lower caloric need. One therefore often sees overweight if these foods are given when the child is at home. The lactic acid foods seem to be of value in the presence of numerous infections. It seems that if the children are placed on this food, they show less parenteral dyspepsia. Naturally, any lactic acid mixture must contain sufficient carbohydrates and fats; however, if any nutritional disturbance arises, the high fat and sugar can be dangerous. Kleinschmidt therefore believed that his butter flour mixture is of great practical

value, since here the fat and carbohydrate content can be quickly changed without changing the food. He also believed that his use of semisolid foods early, that is, in the fourth and fifth months, and the sole use of such foods without liquid milk at the end of the year, is of value in preventing the development of overly fat children.

THE DIFFICULTIES OF JUDGING THE ABILITY TO NURSE. MICHAEL COHN, Gesundh. f. d. Kindesalter **2**:337 (June) 1927.

Owing to the granting of premiums to nursing mothers, the physicians in Germany are under the necessity of determining whether an applicant is actually nursing her child. Cohn discusses the difficulty and shows that there is no absolute method of determining this with the possible exception of the use of the scales before and after nursing. If expression yields absolutely no milk, the result can be interpreted only in one way. However, if a few drops are obtained, the decision is already in doubt. Since often a breast can yield milk only to the nursing child the colostrum is also difficult to judge. Naturally, if the colostrum is apparent, it speaks only for a child that is being weaned. However, a nursing breast may contain milk which yields a few colostral bodies. In weaning, the milk gradually develops a yellowish color, and toward the end it becomes of a watery cloudy character when the fat entirely disappears. The increase in viscosity is also of value. During weaning the drops of milk take on an adhesive quality, a characteristic that can be tested by pulling a drop apart between two fingers. Cohn believes that it takes about fourteen days for the milk to develop colostral characteristics.

REICHLE, Cleveland.

INDICATIONS FOR A MILK-FREE DIET IN INFANTS AND YOUNG CHILDREN. A. POPOWA, Monatschr. f. Kinderh. **35**:416, 1927.

The author decided to do a little more work with the diet of almond milk described by Moll in 1923. His formula is prepared by grinding up 150 Gm. of sweet almonds in 1 liter of water. This mixture, when strained, contains 400 calories per liter. The whey of milk obtained by adding 4 Gm. of calcium lactate per liter is added in equal parts to the almond milk. Carbohydrate addition consists of 5 per cent rice flour and 10 per cent beet sugar. The finished product is palatable, contains from about 650 to 680 calories, and often can be taken by children for long periods of time.

The author then takes up the question of exudative diathesis. After reviewing the literature, he comes to the conclusion that there is much evidence in favor of Czerny's view that the fat of cow's milk is injurious to these patients. In a study on fifty-six rats fed on cream, butter and cakes, the author believed that exudative skin manifestation occurred in many.

There is, of course, the factor of casein and anaphylaxis to be considered.

Accordingly, the author tried his diet of almond milk in three infants with exudative diathesis. He believes that this diet was beneficial both to the exudative skin manifestations and to the lymphatic hyperplasia.

GERSTLEY, Chicago.

STUDIES IN RICKETS: II. FURTHER COMMUNICATION ON THE QUESTION OF GLYCOLYSIS. JOACHIM BROCK and A. WELCKER, Ztschr. f. Kinderh. **43**:193 (March) 1927.

Blood glycolysis in experimental rickets in rats is just the same as it is in rickets in children, with a low inorganic phosphorus curve which is usually diminished or abolished. A diminution of glycolysis measured by the formation of lactic acid in broth made from the liver could be demonstrated in rachitic animals, indeed, on the average to a half of the normal value. Skeletal muscle in rachitic rats has a normal content of inorganic as well as hexose phosphoric acid.

THE ANALYSIS OF REINDEER MILK AND ITS USE AS A FOOD IN INFANCY. ARVO YLPPÖ, Ztschr. f. Kinderh. **43**:255 (March) 1927.

Reindeer milk is exceedingly rich in fat, albumin and salts: fat, 18.7 per cent; protein, 11.1 per cent; ash, 1.2 per cent (phosphoric acid, 42.3 per cent; calcium, 20.9 per cent, and iron, 0.012 per cent). Ylppö thinks that reindeer milk, which contains six times as much fat and salts as human milk, is too concentrated for an infant. Disturbances of the general condition, atrophy and anemia, which were observed in the Lapp children fed on reindeer milk, may be dependent on harm done partly by desiccation, partly by fat.

DISTURBANCES OF THE CARBOHYDRATE METABOLISM. JOSEF WARKANY, Ztschr. f. Kinderh. **43**:305 (March) 1927.

Warkany reported the case of a boy, aged 3½, who did not give the impression of being sick, but who seemed underdeveloped; the weight was 11.5 Kg.; the sitting height was 49 cm.; he was mentally backward. On the basis of a leukopenia, thrombopenia, the galactose test, the enlarged spleen, urobilinuria and icterus, the diagnosis of cirrhotic splenomegaly was made. After operative removal, the spleen showed chronic enlargement with progressive tuberculosis of the capsule. There was a disturbance of the carbohydrate metabolism, which was dependent on the cirrhotic changes in the liver, a real levulosuria. The blood sugar was increased by peroral administration of levulose, and levulose was demonstrated in the blood. The formation of glycogen was carried on here with difficulty, and it was manifested by the low fasting blood sugar and the increased tendency to the formation of acetone on the one hand and on the other by a high glycemic quotient.

EMERSON, Boston.

ETIOLOGY OF ATROPHIC CONDITIONS IN CHILDREN AND THE RÔLE OF PROTOPLASMIC DISPERSITY IN DIAGNOSIS. K. SVEHLA and C. SAITZ, Cas. lek. cesk. **64**:1760, 1805 and 1847 (Dec.) 1925.

The physiologic processes during life gradually change the dispersity, condensation or state of aggregation of the tissues from a labile to a stable state. For estimating the state of aggregation, the following methods can be used: (1) solubility, (2) concentration of hydrogen ions, (3) viscosity, (4) osmotic pressure, (5) surface tension, (6) endosmosis and cataphoresis and (7) precipitation. Because of the impracticability of taking larger quantities of blood from the small infants, the Wassermann reaction, precipitation of 0.2 serum with 1 cc. distilled water and 0.2 cc. absolute alcohol and the p_H determination according to Michaelis were carried out. Precipitation ran parallel with the Wassermann, Meinicke and Sachs-Georgi reactions. The intensity of precipitation does not, however, have a bearing on the prognosis. In two cases of manifest syphilis in which the precipitation test was negative, all the other tests were also negative. Increased precipitation possibly appears as long as two months before the other reactions become positive and does not disappear as early as the others during treatment. Small provocative doses of medication increase precipitation. The increased precipitation of serum globulin in syphilis is dependent possibly on the OH radical concentration as the p_H is not appreciably changed.

STULIK, Chicago.

Prenatal Conditions

HEMIHYPERTROPHY AND TWINNING. ARNOLD GESELL, Am. J. M. Sc. **173**:542, 1927.

In 1921, the author reported a case of hemihypertrophy with bibliography (*Arch. Neurol. & Psychiat.* **6**:400 [Oct.] 1921). He has now reported a second case. Both the patients are mentally defective. Reexaminations indicate that physical and mental features remain relatively constant. There are now fifty-

three recorded cases altogether; twenty-three were in males and thirty in females; the right side was affected in thirty-five and the left in eighteen; skin complications were present in twenty-four and mental subnormality in eight. Hemihypertrophy is interpreted as a minimal form of twinning. The relatively high incidence of mental defect probably would have been found still higher if more and careful examinations had been made. The mental defect is ascribed to disturbance of normal development connected with twinning. A supplementary bibliography is given.

ARCH. PATH.

THE UNBORN BABE. CALVIN R. HANNAH, Texas State J. Med. **23**:220 (July) 1927.

Statistics show that of every 1,000,000 births, approximately 3,000 infants will die during the first year from congenital malformations. A small percentage of these defective children may continue to live for a few years and are often mentally defective as well as physically helpless. Most human beings are born physically fit and have certain definite mental potentialities and die because of external causes.

The offspring of healthy parents will usually have in their stream of human protoplasm those properties which will make for health, energy and sanity. There is a normal rate of growth from the time the ovum and spermatozoon unite. The ovum and spermatozoon must be healthy in order that the embryo may develop without malformation. The fertilized ovum is protoplasm evolved to a point that has within it the structural foundation of an infant. The embryo receives its nourishment and transforms it by oxidation into new tissues. Psychic influences probably do not act directly on either the ovum or the spermatozoon. Focal infections may cause cleft palate, deafness, mutism, hydrocephalus, spina bifida and other abnormalities. Internal secretions probably affect the growth of the embryo. Acute and chronic infectious diseases in the mother will claim many an unborn infant.

Precipitated labors are hazardous for the infant and are detrimental to the mother, and are the frequent cause of injury of the brain in the newly born. The child should never be delivered through an undilated cervix, nor should the second stage of labor be hastened unless the condition of the mother or infant demands it. The patient should receive relief from pain. Physicians are vitally interested in the prevention of diseases which would retard the development of the embryo and for its protection in utero. The unborn infant cannot change its physical form and mental characteristics in utero. Efficiency in obstetrics will reduce injuries at birth, and will give to the unborn infant the advantages to which it is honestly entitled—a healthy body and a healthy mind.

MOORE, Dallas.

THE EPITHELIAL HETEROTOPIAS OF THE ALIMENTARY TRACT. A. L. TAYLOR, J. Path. & Bact. **30**:415, 1927.

Epithelial heterotopia of congenital origin occurs with considerable frequency in the alimentary tract. It is limited almost without exception to that portion of the intestine which lies above the level of the ileocecal valve. The superficial congenital heterotopias consist in the substitution of a portion of the normal mucous membrane by areas of mucous membrane foreign to that part, but normal to some other intestinal region. They invariably show an adult structure, the only anomaly being one of situation. The deep congenital heterotopias may occupy any portion of the thickness of the bowel wall and may produce tumor-like structures of characteristic naked-eye appearance. Microscopically, they possess a varied structure, the most frequent constituent being pancreatic tissue. In all cases they contain only those types of cell which are normally found in some region of the alimentary tract. In some cases, as in adenomyoma, they show a marked lack of differentiation, with

evidence of neoplastic activity. The clinical significance of the congenital heterotopias is generally small. The great majority are found by accident at routine postmortem examinations. Rare exceptions to this rule are provided by:

(*a*) The superficial heterotopias of the gastric mucosa in the small intestine and in Meckel's diverticulum, which may be the seat of ulceration with intestinal hemorrhage or perforation.

(*b*) Certain deep heterotopias of the stomach and small intestine, notably the pyloric adenomyomas. These tumors may produce symptoms suggestive either of gastroduodenal ulceration or cancer, and for which operation may be performed.

(*c*) Nodules of aberrant pancreas at the apex of Meckel's diverticulum which may produce mechanical obstruction of the intestine.

(*d*) Certain umbilical polyps, which may cause discomfort or pain and an irritating discharge.

The congenital heterotopias play no part in the production of malignant disease. For this reason local excision only should be practiced in all cases requiring operative treatment. Congenital heterotopia is a dysontogenetic and not an atavistic condition. It is a tissue malformation arising from an error of development of the fetal entoderm. In their distribution and in their antimesenteric situation, the congenital heterotopias correspond closely with the diverticula occurring in the intestine of the embryo. It is concluded that they arise from those embryonic diverticula which abnormally persist. The precise nature of the abnormal stimuli which result in their formation is unknown. The superficial heterotopias of acquired origin occur chiefly in the stomach and consist of epithelium of intestinal type. They are produced by an error of differentiation in the regenerative process accompanying ulcerative lesions. They spring from the "indifferent" cells normally present, when under abnormal stimulation there is a reappearance of an alternative cell character which was previously latent. The deep heterotopias of acquired origin may be of hyperplastic or regenerative type. The hyperplastic type is a rare condition occurring in the stomach by glandular penetration through the vascular gaps in the muscularis mucosae. The regenerative type is found in all parts of the alimentary tract in ulcerative lesions where the muscularis mucosa is destroyed. It consists of glandular structures segregated in the submucous tissues during the regenerative process. Evidence is brought forward that the growth of these heterotopias though abnormal in situation is of typical character. Both superficial and deep heterotopias of acquired origin consist of fully differentiated or normally developing tissue. They may coexist with carcinoma but they take no part in its production.

AUTHOR'S SUMMARY.

PATHOGENESIS OF CYSTIC LIVER. L. WACKERLIE, Virchows Arch. f. path. Anat. **262**:508, 1926.

In two still-born fetuses, a moderate grade of cystic liver was associated with cystic kidneys and other maldevelopments, and in a woman, aged 56, a more marked degree of formation of cysts in the liver was associated with bilateral cystic kidneys, which led to death in uremia. In the three livers, an excess formation of interlobular stroma had taken place, and at the periphery of the interlobular fields excessive numbers of small bile ducts lined by cuboidal epithelium were present. It was ducts of the latter groups which had become cystically dilated. The connection of peripheral interlobular ducts and cysts with intralobular bile canaliculi could be seen, but it was impossible to detect any communication of the cystic peripheral ducts with the main interlobular ducts. Wackerle accepts the independent formation during embryonic development of the intralobular bile canaliculi and the extrahepatic ducts system together with the interlobular ducts. The union of the two systems occurs in the peripheral one of the interlobular tissue, the smaller peripheral ducts in this zone in his cases belonging to the intralobular system. The process leading

to cystic liver is looked on as a hamartoma, in the sense of Albrecht, which manifests itself by a disproportionate formation of interlobular mesenchymal tissue. The intralobular ducts are unable to penetrate this tissue, and hence fail to accomplish their union with the main interlobular ducts.

PLACENTAL TRANSMISSION OF HODGKIN'S DISEASE. A. PRIESEL and A. WINKELBAUER, Virchows Arch. f. path. Anat. **262**:749, 1926.

Two weeks before the end of a normal pregnancy, painless enlargements of the superficial lymph nodes developed in a woman, aged 31. From the microscopic examination of an excised node, the diagnosis of lymphogranulomatosis was made. A nodular swelling was noted in the left supra-orbital region of the child when the latter was 11 weeks old; the mother had died in the meantime and a necropsy had not been performed. The child died at the age of 4½ months. The characteristic histopathologic changes of lymphogranulomatosis were present in the bones of the skull, the bone marrow, the liver, the thymus and the retroperitoneal and mesenteric lymph nodes. The early onset of the disease in the child convinces the authors of the placental transmission of the process. Bacteriologic examinations and animal inoculations were negative for tuberculosis.

CONGENITAL DIVERTICULUM OF THE URINARY BLADDER. S. USAMI, Virchows Arch. f. path. Anat. **263**:99, 1927.

Usami makes a case of diverticulum of the urinary bladder in an adult the basis of a discussion of the differences between acquired and congenital diverticula. The latter are divided into those that are present at birth and those that develop later on the basis of a congenital factor, one of the commonest of which is constituted by accessory embryonic ureteral remnants within the bladder wall. In Usami's case there was a large diverticulum occupied by a stone, three smaller intramural diverticula and numerous areas in which the mucosa dipped down between the muscle bundles. Urinary retention was not present to explain the conditions seen, which were believed to be the result of a developmental anomaly of the bladder. In support of this view was the presence within one intramural ureteral sheath of a small, epithelial lined ductlike passage, which was interpreted as a rudimentary accessory ureter.

TRANSPOSITION OF AORTA AND PULMONARY ARTERY. F. HOGENAUER, Virchows Arch. f. path. Anat. **263**:174, 1927.

An example of transposition of the aortic and pulmonic trunks of the heart of a child who died at the age of 2 months is explained on the basis of Spitzer's theory of incomplete torsion of the primitive cardiovascular tube, with persistence of the phylogenetic right-sided aorta.

O. T. SCHULTZ.

A NEW SIGN OF PREMATURITY. DIVISION OF THE BODY INTO TWO HALVES OF DIFFERENT COLOR. L. O. FINKELSTEIN and R. A. WILFAND, Ztschr. f. Kinderh. **43**:201 (March) 1927.

Finkelstein and Wilfand described a new sign of prematurity observed by them and reported four cases in which the body in the lateral recumbent position showed two sharply symmetrical halves of different color. This phenomenon lasted from two to two and one-half months. The underlying half was red, tinged with blue; the upper half was pale or white; the line between the halves was precisely the median line, and there was no gradual transition from one color to the other. By turning the child over to the other side, the picture was reversed. The authors assumed the cause to be a disturbance of the connections between the vasomotor innervation of the two halves of the body and that the site of the disturbance is perhaps in the corpora striata; that is, the vasomotor center is a double one.

EMERSON, Boston.

EARLY EMBRYONIC CAUSES OF MALFORMATIONS OF THE NERVOUS SYSTEM. ERNST DE VRIES, Schweiz. Arch. f. Neurol. u. Psychiat. **19**:274, 1926.

Earlier investigations have taught us a great deal concerning the causes of malformations of the central nervous system. Much, however, remains unknown. The attempt to attribute status verrucosis deformis (Ranke) of the cerebrum and certain heterotavias of the cerebellum to pathologic processes of the membranes seems justified.

De Vries reported a number of cases. The first was that of a human fetus of 9 months, with polygyria of the cerebellum. Important observations were that the polygyria involved the three outer layers only, and that the leptomeninges in the sulci of the diseased areas were absent. As a result, the adjacent structures became fused. De Vries believes that an early inflammation of the membranes took place; this left a thickened pia, as a result of which this membrane could not keep pace with the formation of the convolutions, which, in turn, resulted in adhesions between the convolutions and prevented the formation of normal secondary convolutions. Over the occipital lobe there was a definite meningitis with thickening and infiltration of the leptomeninges. The underlying structures were involved secondarily, probably due to ischemia.

Case 2 was one of heterotopia and heterotaxia of the cerebellum. The patient was a simple-minded girl who died at the age of 17 years. There had been prominent cerebellar symptoms but no history of previous disease of the brain. Anatomically there was slight microcephaly, simple type of cerebral convolutions, marked atrophy or agenesis of the cerebellum and doubling of the granular layer. In the vermis there were two symmetrically placed nodules of gray matter the size of a pea. Whether the picture produced was traumatic or meningitic could not be determined.

Case 3 was one of status verrucosis deformis of the cerebrum and extensive heterotaxia of the cerebellum. The child had been an idiot and had died at the age of 9 years. As an infant, he had apparently been well, and only gradually did the parents become aware of the mental deficiency. There had been slight convulsions, and he had had an attack of inflammation of the lungs. His movements had been extremely spastic and ataxic, the extremities had become spastic, and the posture had been one of extreme opisthotonos. The cerebellum showed a large number of heterotactic areas. Investigation of these revealed the interesting fact that there was no pia between the convolutions, and that these were intimately fused. In this vicinity, there was no evidence of an early meningitis. Examination of the remaining structures showed some scar formation in the cerebellum, heterotopia of the Purkinje cells, thickening of the pia covering the cerebrum with marked clouding at the tips of the temporal lobes, enlargement of the ventricles, microgyria and polygyria of the cerebrum, the so-called status verrucosis deformis, and an excessive folding in the region of the external geniculate body.

The fourth case was one of status verrucosis deformis of one hemisphere. The patient had been epileptic and feebleminded since childhood. There was no history of encephalitis. There had been a spastic infantile hemiplegia on the right side. He died at the age of 54 of tuberculosis, without evidence of meningitis. At necropsy there was an accumulation of pus over both hemispheres. The convexity of the left hemisphere showed a typical microgyria. This whole hemisphere was small and the opercular formation was only indicated. In the region of the microgyria, the three outer layers showed excessive folding, whereas the two deeper layers showed the normal convolutional structure. The pia was seen to extend into the principal sulci; however, it did not extend into the deformed sulci.

It would seem from these observations that the formation of the gyri and sulci cannot progress far when the pia fails to follow. In contradistinction to Brun, who attributed many forms of malformation to a toxic endogenous cause, De Vries believes that at least one type may be explained on a mechanical basis and is a result of disease of the meninges.

WOLTMAN, Rochester, Minn.

Diseases of the New-Born

A Study of Behavior in the New-Born. Louise Taylor-Jones, Am. J. M. Sc. **174**:377 (Sept.) 1927.

In a record of the reaction of seventy-five healthy infants to certain external stimuli during the first twenty-four hours of life and the same reaction to the same stimuli during the second twenty-four hours of life, Dr. Jones was able to demonstrate by certain tests made on the day of birth that the infant has many activities, individualities and personality. Infants learn the first day of life, so that the formation of habits and character begins then. The study also showed that most of the special senses are used immediately after birth. Dr. Jones found that during the physical examination measurements of the arms and legs are the same, except in infants born before term the arm was longer, and if the legs were longer, the infant had arrived after the expected date.

Henske, Omaha.

Paralysis of the Right Diaphragm in the New-Born Due to Phrenic Nerve Injury. James E. Dyson, J. A. M. A. **88**:94 (Jan. 8) 1927.

An infant, aged 6 weeks, was found to have a paralysis of the right diaphragm apparently following accidental puncture of the neck in the region of the scalene muscle and the phrenic nerve with scissors at the time of birth. The patient made a complete recovery in six months.

Bonar, Salt Lake City.

Cerebromeningeal Lesions of the New-Born Dependent on Labor. R. Waitz, Rev. franç. de pediat. **3**:434, 1927.

Waitz based his article on a careful study of the brain and meninges in forty-four patients, six dying during labor and thirty-eight, during the first fifteen days of life. The following lesions were found: contusions and elongations, extradural hematomas, vasodilatations, edema and hemorrhages. The first are due to trauma and are rare, except for the extradural hematomas of the spine, which are without serious import. In contrast, the last three lesions (vasodilatations, edema and hemorrhages), which are directly linked up with labor, are important. They are found in the subarachnoid space, in the parenchyma of the nervous tissue and in the neighborhood of the ventricles and the choroid plexus. Acute edema of the meninges may be present. The evolution of the lesions varies, depending on the severity and the type of tissue involved. The sequelae are of four types: albuminous pachymeningitis, suppression of the blood supply, destruction of the parenchyma and lesions of the choroid plexus. It would seem that these sequelae may play a major rôle in the genesis of obstetric encephalopathies and of certain hydrocephalies.

Leslie, Chicago.

The Cause of Death in the New-Born. H. Hook, Beitr. z. path. Anat. u. z. allg. Path. **77**:456, 1927.

Hook shows that in addition to cerebral hemorrhage, infection, especially a confluent pneumonia following the aspiration of infected amniotic fluid, is an important factor in the death of new-born babies. This conclusion is based on the observations from numerous necropsies on new-born infants. His series of cranial hemorrhages is divided into those with definite evidence of trauma from birth, as qualified by injuries to the scalp, cephalhematoma and tears into the cerebellar tentorium; and those not due to external trauma, in which the foregoing qualifications are missing. The majority of his cases fall into the latter group. Hemorrhage in these cases is not considered the essential cause of death but a preagonal or agonal manifestation. The external mechanical stress of birth in a condition of circulatory stasis, or a toxic infectious factor,

plays an important rôle in causing nontraumatic hemorrhage. In support of this view are two infants whose mothers died of gas poisoning and heart failure due to endocarditis before labor began; both infants had cranial hemorrhages. The presence and significance of fatty granules in and about the cells of the glia are discussed at length. This process was minimal when vascular stasis was marked. The association of this condition with infections at distant foci was noted in fifteen cases, in eight of which cerebral hemorrhage did not occur. Fatty changes are conceded to be a sign of fatal trauma from birth, if etiologic factors other than hemorrhage can be ruled out, and if the localization is in a region of the brain that might undeniably cause death. Antenatal aspiration was noted in forty-three of fifty-four lungs examined. A focal confluent pneumonia followed, especially in cases associated with ruptured membranes over a long period, and when the mother was suffering from intrapartum fever. When the pulmonary reactions were slight, the cranial hemorrhage seemed to point definitely to the cause of death. Hook's work indicates that despite the cranial observations, the examination of other organs may be necessary to ascertain the cause of death.

A. J. Kobak.

Developmental Anomalies of Internal Organs from the Standpoint of Diagnosis and Viability of the New-Born. J. Simecek Vitkovice, Čas. lék. česk. **64**:1874 (Dec.) 1925.

The author mentioned diaphragmatic hernia and congenital heart lesion as causes of death in the new-born, which apparently produce no effects during pregnancy or during severe labor. Severe hernia is likely to be associated with pulmonary aplasia, and convulsive attempts at inspiration seems to be the typical sequel. This is in contrast to the temporary cyanosis and respiratory difficulty in obstruction due to mucus or other extraneous substance. Cyanosis, with spasmodic attempts at inspiration in the presence of normal heart action, is diagnostic of massive diaphragmatic hernia. Since cardiac murmurs are not always present even in severe congenital cardiac lesions, cyanosis, rapid respiration, drowsiness, apathy, poor nursing with shortness of breath and early signs of cardiac decompensation, such as edema and epistaxis, must be relied on for diagnosis.

Stulik, Chicago.

Acute Infections

The Bacteriology of Whooping Cough. George M. Lawson and Mary Muether, J. A. M. A. **89**:275 (July 23) 1927.

Cultures taken from patients with whooping cough are an added aid in the early diagnosis of the disease in carriers, second cases and suspected cases, all of which may never be detected but which may be diagnosed by bacteriologic studies. During the catarrhal stage, positive cultures are obtained with considerable regularity, but as the disease progresses, there is a coincident decline in the percentage of positive cultures. The requirements for quarantine should include the catarrhal stage of the disease, and termination should be determined culturally.

Bonar, Salt Lake City.

Ocular Complications of Mumps. Villard, Arch. d'opht. **4**:492, 1927.

The best known complications of mumps are those affecting the lacrimal gland, the dacryo-adenitis of mumps, in which the lacrimal gland is affected in the same way as the parotid gland. It usually occurs after the parotitis has developed, but, exceptionally, swelling of the lacrimal gland may precede the parotitis by several days. The swelling, pain and lacrimination require from several days to one or two weeks to subside. Three cases have been reported in which both lacrimal glands were involved during an epidemic of mumps without any involvement of the parotid. In all cases the inflammation

subsided without damage to the eye and did not require any special treatment except hot applications. An optic neuritis occurs about as frequently as adenitis. This usually appears in the form of neuroretinitis with a typical fundus picture of hyperemia of the disk with edema of the surrounding retina. In a few cases, vision has been considerably and permanently reduced. Villard reports three cases of a less uncommon complication, involvement of the uveal tract. Two of his cases showed mild iritis and one iridocyclitis with numerous deposits on the cornea. All subsided rapidly without other treatment than atropine, and normal vision was obtained. A rare complication, acute purulent dacryocystitis, is illustrated by another of the author's cases. The literature is reviewed and a bibliography is included.

GIFFORD, Omaha.

TREATMENT WITH PREPARATION OF NEUCLEINS AND PHENOL IN PEDIATRICS; NONSPECIFIC PROTEIN THERAPY. BORTOLUCCI PIER GUISEPPE, Riv. di clin. pediat. **25**:578, 1927.

The author injected a preparation of nucleins and phenol intramuscularly each day into 243 patients with acute infectious diseases, such as measles, mumps, rheumatic fever, pneumonia, erysipelas and typhoid fever. He did not give detailed cases reports, but claimed excellent results from this nonspecific protein therapy.

HIGGINS, Boston.

PERTUSSIS. T. SZONTAGH, Jahrb. f. Kinderh. **111**:185 (Jan.) 1926.

Pertussis is periodically epidemic, and epidemics of measles tend to precede or follow epidemics of v. pertussis. Szontagh considers pertussis a symptom of a neuroarthritic diathesis and a disease in which catarrhal symptoms alternate with nervous symptoms. The rhythmic onset of the paroxysms suggests a toxic origin. The author says that pertussis is not contagious in contrast to measles. He has never seen one child infect another. Once all the children in one of his wards developed whooping cough simultaneously, but none of the other wards were affected.

EHRMANN, Chicago.

SECOND ILLNESS IN MEASLES. ADOLF F. HECHT, Ztschr. f. Kinderh. **43**:149 (Feb.) 1927.

By second illness in measles is meant a recurrence of fever, which was observed in eighty-two cases; twenty-five times on the ninth day and thirty-nine times on the eighth, tenth and eleventh days after the breaking out of the exanthem. Second illness is not an after-sickness but is a result of the redevelopment of the capacity for reaction after the anergic period characteristic of measles has been overcome. Support for this point of view is the occurrence of an anaphylactic outbreak on the tenth day of erythema and a residual exanthem on the eighteenth day of erythema (third illness).

PAUL W. EMERSON, Boston.

CRITICISM OF THE DICK CONCEPTION OF SCARLET FEVER BASED ON CLINICAL OBSERVATIONS AND RESULTS. SELMA MEYER, Ztschr. f. Kinderh. **43**:258, 1927.

From a study of the literature and from the author's researches, the conflicting results obtained with the Dick reaction in patients with scarlet fever, in those who have had scarlet fever, and in those who have never had scarlet fever lead to the conclusion that this reaction is not the expression of susceptibility or immunity to scarlet fever but is only an indication of existing immunity to an infection with streptococci. The several arguments for the conception of scarlet fever held by the Dicks, the specificity of scarlet fever streptococci and their toxin, the neutralization of the position with the serum of patients convalescing from scarlet fever and of those giving negative reactions to the Dick test, the power of this serum and of scarlet fever horse serum

to bleach the exanthum, the curative value of scarlet fever serum and, finally, the production of symptoms resembling scarlet fever, and the immunization against scarlet fever by means of the toxin of streptococci, all these bits of evidence lose some of their importance if one considers them critically and judges them by experimental results. They are surely evidence only for the formation of the toxin and the antitoxin of streptococci. With the toxin of other and different streptococci, reactions analogous to the Dick reaction can be obtained.

The curative serum for scarlet fever and likewise the serum from convalescent patients has just as good a detoxifying action in other streptococcic infections such as puerperal fever, phlegmon, erysipelas and empyema as it has in scarlet fever. According to the anaphylactic hypothesis, as a result of the common infections with streptococci, the broken down products of the bacteria sensitize by the formation of streptococcic antibodies against renewed infection with streptococci and suddenly produce the anaphylactic symptom complex, scarlet fever. By this theory the differences of experimental results with Dick toxin may be smoothed out, the contradictions of the Dick hypothesis cleared away and a correct conception provided for the origin of scarlet fever in accordance with the facts.

A New Method of Treating Tetanus. E. Rychlik, Čas. lék. česk. **64**:1524 (Oct.) 1925.

In four cases in which the treatment consisted of intraspinal injection of procaine-antitoxin mixtures, a rapid cessation of convulsions resulted, but in three cases reappeared at intervals. In a moribund patient with cyanosis 5 cc. of antitoxin and 10 cc. of 0.5 per cent procaine hydrochloride was injected subdurally through an opening made in the parietal bone. This produced a general cessation of convulsions associated with motor aphasia lasting four hours. Subsequent postmortem examination showed neither injury to the brain nor hemorrhagic spots which could be said to be due to the procedure. Fifteen cubic centimeters of a 0.5 per cent solution of procaine hydrochloride injected into the masseter muscle overcame a severe trismus, and the patient was able to eat in comfort. Paravertebral injection was painful, and repeated injections aggravated the opisthotonus. This was controlled in the general relaxation following lumbar injection. Perhaps the procaine hinders the combining of the nerve and toxin elements (specific). The author recommends an initial intraspinal injection of 0.12 Gm. procaine hydrochloride and 3 cc. antitoxin after removal of an appreciable amount of spinal fluid to aid in the intimacy between the mixture and the nerve elements (cerebrospinal structures). In cephalic tetanus, before involvement of the fourth ventricle, intracranial injection is recommended for trial. The question of the variability of the cerebrospinal structures to lumbar anesthesia is left open.

Stulik, Chicago.

Chronic Infections

Tuberculosis Case Finding in Children. Henry D. Chadwick, Am. Rev. Tuberc. **15**:601, 1927.

About 30,000 children from the grade schools in Massachusetts were examined for evidences of tuberculosis. They were selected from the bulk of the school population on account of underweight, a history of contact or because they were known to be in poor health. A positive Pirquet test was obtained in 2,806 of those tested. Based on careful physical and roentgen-ray examinations and cutaneous tests, a diagnosis of pulmonary tuberculosis was made in 45 instances, hilum tuberculosis in 1,106 and 2,656 of the children were classified as suspects.

The author concludes that in children the usual examination of the chest is not to be depended on in making a diagnosis of tuberculosis. The physical

signs in hilum tuberculosis are slight and inconclusive. Râles are seldom found in pulmonary tuberculosis except in advanced cases, and are usually due to some other cause than tuberculosis. The best procedure is to make cutaneous or intracutaneous tests on all children, and to make a roentgen-ray examination of all reactors. By these means, latent as well as active forms will be brought to light. This is important since latency in children is only relative, as not enough time can have elapsed for the foci of the disease to have become thoroughly sclerosed or calcified, and reactivation may occur at any time.

SMITH, Ogden, Utah.

HEREDITARY SYPHILIS IN THE FOUNDLING AND SOME NOTES ON ITS TREATMENT. GUIDO GUIDI, Riv. di clin. pediat. **25**:570, 1927.

In Italy, the death rate of illegitimate children is approximately eight times that of legitimate children during the first year of life.

In an orphanage under the author's supervision from 1923 to 1925, there were 1,150 admissions: forty-four were children of syphilitic parentage; twenty-five of these did not show symptoms of the disease and had a negative Wassermann reaction; nineteen showed symptoms and had a positive Wassermann reaction, four dying from the disease.

The author cited a case in which the infant of a syphilitic mother showed no symptoms of syphilis and had a negative Wassermann reaction. Later, it developed severe symptoms. The author advised that antisyphilitic treatment be given to all infants with a family history of syphilis, regardless of the absence of symptoms or a negative Wassermann reaction.

HIGGINS, Boston.

TUBERCULOSIS SOCIAL SERVICE FOR CHILDREN. JULIUS PEISER, Gesundh. f. d. Kindesalter **2**:297 (June) 1927.

The article is concerned largely with the discussion of the source and manner of tuberculous contagion and with its statistics. In Berlin, the children who come to the social service bureaus are either invited by the social workers or by the school physician, chiefly because of the presence of tuberculosis in the family or because the general condition of the child arouses suspicion. In determining the activity of tuberculosis, Peiser did not find the Wassermann tuberculosis reaction of any value. He lays more stress on the differential diagnosis according to Schilling and to the sedimentation test. To him, the most valuable aid in prophylaxis is the vacation colonies. The children are chosen from environments which threatened contagion, and an attempt is not made to choose only such children as are below the normal standards in physical constitution. As an indication of the value of the procedure, the body weight is not regarded with much favor. Skin tonus, increase in hemoglobin and in the subcutaneous fat fold are regarded of great value. The decrease in the abdominal circumference as an indication of the increase in muscle tone is also praised. Pigmentation is desirable, as is also the development of deeper sleep and better appetite. In Stettin, 3.7 times as many infections occur in households in which prophylactic treatment for tuberculosis had not been given as in those in which it had been given.

REICHLE, Cleveland.

Internal Diseases

BONE DEVELOPMENT IN DIABETIC CHILDREN: A ROENTGEN STUDY. L. B. MORRISON and I. K. BOGAN, Am. J. M. Sc. **174**:313 (Sept.) 1927.

Morrison and Bogan made a roentgenologic study of sixty-eight diabetic children under 15 years of age. They demonstrated that development of bone is in advance of the chronologic age in children with diabetes of recent onset, and that the reverse occurs in diabetes of long standing. Narrowness of the shaft and thinness of the cortex characterize the diabetes of long

standing in children. Atrophy of the bone occurs in a certain number of children who develop diabetes before the ninth year, but not above that age.

HENSKE, Omaha.

SUBACUTE INTESTINAL OBSTRUCTION WITH UNUSUAL DILATATION OF THE COLON SHOWN ROENTGENOLOGICALLY. EMANUEL W. LIPSCHUTZ, Am. J. Roentgol. **15**:326 (April) 1926.

An enlargement of the colon was diagnosed merely from its contained air, and an opaque substance was not necessary. On operation it was found that a portion of the pelvic colon was adherent in the scrotum. This was differentiated from the Hirshsprung's disease on the basis of the history of recent onset in an adult.

MYOSITIS OSSIFICANS PROGRESSIVA: CASE REPORT AND REVIEW OF THE LITERATURE. L. FREELAND MAGRUDER, Am. J. Roentgenol. **15**:328 (April) 1926.

Roentgen-ray examination in a man, aged 22, showed a shaft of bone extending from the external occipital protuberance and corresponding to the ligamentum nuchae. Both scapulae were fixed by bone shafts extending to the ribs. The shaft from the occiput extended to the sacrum. New bone was also present in the right elbow, both hips, both feet, the thighs and legs. Removal of bone and implantation of radium salts is thought justifiable by the author with the idea of inhibiting osteogenesis and producing endarteritis of the small blood vessels. Reports of 133 cases were found in the literature, six of which were thought to be congenital; twenty-nine were in infants under 2 and forty in children under 5 years, after which the case incidence somewhat decreases. Ossification in the intramuscular connective tissue, fascia and aponeuroses following capillary hemorrhage may be the cause. Microscopically, myositis ossificans traumatica and myositis ossificans progressiva are identical. Brachydactylia, brachyphalangia or brachydactyphalangia are present in all true cases. Possibly the microdactylia, being of endogenic origin has a bearing on imperfect terminal vascular development and the hemorrhage.

STULIK, Chicago.

CLINICAL MANIFESTATIONS OF AN ENLARGED THYMUS. E. A. MORGAN, G. H. ROLPH and ALAN BROWN, J. A. M. A. **88**:703 (March 5) 1927.

The authors review fifty-four cases of enlarged thymus, two of which were discovered accidentally. The clinical manifestations described were: breath-holding spasms in twenty-nine; syncope in nine; cyanosis in eight and cough in seven. Persistent hoarse cough was the chief complaint in four, while in the remainder it was a secondary complaint. Choking attacks occurred in five cases, and noisy nasal breathing in six cases. Typical thymic asthma was present in only three cases. Nervous manifestations such as sleeplessness, restlessness and irritability were reported in three instances to have disappeared following roentgen-ray therapy. Twelve, or 22 per cent, of the patients suffered from either local or general eczema. Roentgen-ray therapy proved efficacious in relieving symptoms in 96 per cent of the cases. Recurrences of thymic enlargement or of clinical manifestations were noted in twelve patients, all of whom were given further treatment with resulting complete recovery. If not carried to excess, roentgen-ray treatment is unattended by danger.

EPHEDRINE IN BRONCHIAL ASTHMA OF CHILDREN. G. F. MUNNS and C. A. ALDRICH, J. A. M. A. **88**:1233 (April 16) 1927.

Twelve of twenty-two children with bronchial asthma were relieved within from thirty to forty-five minutes by the oral administration of ephedrine sulphate. Four patients were only partly relieved, and in six instances the drug had no apparent beneficial effect. Eight of the nine patients with per-

sistent cough as a major symptom were effectively relieved. Ephedrine seems to be a valuable remedy in many instances of asthma, and especially in the milder cases as a method of relieving cough.

RUPTURED GASTRIC ULCER IN INFANCY. H. E. BULKA, J. A. M. A. **89**:198 (July 16) 1927.

An autopsy performed on an infant, aged 6 days, revealed: a perforated gastric ulcer, acute peritonitis, gastric hemorrhage, pneumoperitoneum and partial atelectasis of both lungs.

THYMIC STRIDOR. W. WALTER WASSON, J. A. M. A. **89**:1025 (Sept. 24) 1927.

The author is of the opinion that it is unfair to assume that a case of stridor is due entirely to the thymus, based on roentgenologic evidence of a large thymus or even of a small thymus, thus depriving the infant of further examination directed toward the sinuses or the lungs. Certainly the respiratory tract is often involved in this class of cases with or without association with an enlarged thymus. Methods directed toward relieving infection have relieved the respiratory stridor in a number of cases which did not appear any different from those usually diagnosed as thymic stridor. Wasson has not observed a change in the stridor with decrease or increase in the size of the thymus. Radiation therapy may be the treatment of choice when the mother will not cooperate in other forms of treatment or in neglected cases.

BONAR, Salt Lake City.

GIARDIASIS IN AN INFANT: INFECTIONS BY THE TRICHOMONAS HOMINIS; INFANTILE ECLAMPSIA; EXUDATIVE ENTERITIS. JOHN ZAHORSKY, M. Clin. N. Amer. **11**:311 (Sept.) 1927.

Zahorsky insists on the value of microscopic examination of the stools in diarrheal conditions in infancy. Significant observations are mucous curds, mucous plaques, ova and protozoa and cells, especially leukocytes and macrophages. Cases of infestation with *Giardia* and *Trichomonas hominis*, infantile eclampsia due to enteritis and exudative enteritis are detailed, in which such examinations were of diagnostic and therapeutic value. Zahorsky says that it is a mistake to ascribe a nutritional disorder to parenteral infection without excluding enteral infection by examination of the stool.

PURE CONGENITAL DEXTROCARDIA. ALBERT E. TAUSSIG, M. Clin. N. Amer. **11**:351 (Sept.) 1927.

Two varieties of pure congenital dextrocardia occur. In the commoner form, the heart occupies about the same position that it does in early fetal life, almost symmetrically in the chest, with the left chambers to the left and the right to the right of the midline. Later, the apical portion grows rapidly and swings to the left. Occasionally, the apex swings to the right producing dextrocardia. The right chambers, however, continue to receive the blood from the peripheral circulation and the left that from the lungs. The electrocardiogram is that of the normal heart. Other congenital defects frequently accompany this condition, such as patent ductus Botalli and open foramen ovale. In the other rarer form, there is a true mirror image of the normal heart, with the chambers occupying the left side receiving the blood from the peripheral circulation and those on the right, the blood from the lungs. The electrocardiogram in this variety is reversed.

INFANTILE DEFECTIVENESS. WILLIAM ENGELBACH, M. Clin. N. Amer. **11**:381 (Sept.) 1927.

Engelbach states that the diagnosis of mental aberrancy in children is usually deducible from the physical signs during the first year of life. Indeed,

a prenatal estimation of embryonal development from a thyroid standpoint is possible through observation of the gravid woman. Symptoms in the latter, suggestive of possible thyroid deficiency in the fetus, are lack of increase in the metabolic rate and of the normal slight transient enlargement of the thyroid. In the infant, the hypothyroid state is first indicated by overweight and delayed appearance of the osseous nuclei of the distal epiphysis of the femur, the proximal epiphysis of the tibia and of two tarsal bones, talus and calcaneus, all of which should be present at birth. Other significant symptoms which may be detected during the first year are delayed appearance of additional osseous nuclei, late separation of the umbilical cord, with or without infection, delayed eruption of teeth and late walking and talking. Parathyroid deficiency manifests itself in increased irritability, spasmophilia, laryngismus stridulus, infantile tetany and spasms. Failure of the posterior lobe of the pituitary gland to supply the normal amount of its specific hormone leads to juvenile adiposity. This condition rarely appears before the fifth or sixth year, and aside from increased weight, usually causes few symptoms before adolescence.

The author emphasizes the importance of recognizing these and other deficiencies of the ductless glands in children in their initial stages, when in many instances they may be successfully treated.

THE SYSTEMIC MANIFESTATIONS OF UPPER RESPIRATORY INFECTIONS. BORDEN S. VEEDER, M. Clin. N. Amer. **11**:415 (Sept.) 1927.

The seriousness of the "common cold" in infants and children is not to be judged solely by its local manifestations, but by its systemic reactions as well. It is even more important to recognize the causal relationship that frequently exists between chronic local infections in the nasopharynx, tonsils and sinuses, and recurrent gastro-intestinal disease, nephritis, arthritis and the like. However, tonsils and adenoids should not be looked on as the cause of all of children's troubles. During infancy, the tonsils rarely give trouble either on account of their size or as the seat of chronic infection. Adenoids at this time, however, are often the source of trouble, and their removal is frequently indicated when the tonsils should be allowed to remain. The chief reason for the removal of tonsils lies in their relation to an active or a potential systemic toxemia, and this should be the point at issue when the question of tonsillectomy comes up. Roentgenograms of the sinuses should be more frequently made in children with obscure systemic toxemias, and the possibility of mastoid infection in persistent gastro-enteritis should be carefully considered.

THYMIC ENLARGEMENT AS A CAUSE OF ATELECTASIS. JOSEPH P. COSTELLO, M. Clin. N. Amer. **11**:583 (Sept.) 1927.

The author's premise is that the thymus, when enlarged at birth, can through pressure prevent the entrance of air into the pulmonary vesicles, resulting in pulmonary atelectasis. This condition is not present at birth, but develops in the course of a few hours. The predominating symptom is rapid, shallow respiration. Nursing is interfered with, and there may be a peculiar short cry. Cyanosis is present or absent, depending on the amount of involvement of the lungs. The infant's color may improve slightly when he is changed from the prone to the sitting position. Aside from the signs of thymic enlargement, physical examination of the chest reveals hyperresonance approaching flatness and distant breath sounds of tubular quality. The abdomen is distended, and the liver and spleen may be enlarged from passive congestion. The roentgen ray is valuable in diagnosis and treatment. On account of the difficulty the infant experiences in nursing, food should be given slowly and in a concentrated form. Dehydration should be prevented. Crying should be induced from time to time to favor expansion of the lungs. Two cases are reported with autopsies.

SMITH, Ogden, Utah.

Testicular Tumors of Congenital Origin. J. Howell Evans, Practitioner **119**:52 (July) 1927.

For years the idea has been advanced that the retained testicle is more liable to malignant disease. It is the author's opinion that the undescent of the testes is frequently due to a congenital aberration. There is a great variety of congenital abnormalities, such as cysts and dermoid tumors, including teratomas.

Many theories regarding the pathologic change have been advanced. Two are particularly open to consideration. The theory of parthenogenesis involves the idea of abnormal proliferative changes occurring either in the spermatozoa or in the primordial ova which may remain in the testes. The blastomeric theory involves the presence of blastomeres which have not gone so far as full differentiation of the fertilized polar bodies.

Malignant to disease may develop in any such tumor formation, resulting in a myxosarcoma or a myxochondrocarcinoma. The chorion epithelioma is blastomeric in origin. Treatment is operative.

Gelston, San Francisco.

Convulsions Following Insulin and the Elaboration of an Insulin That Does Not Cause Convulsions. Free Insulin. Ivo Macela, Čas. lék. česk. **64**:1553 (Oct.) 1925.

Insulin is composed of at least two fractions; one that produces a hypoglycemia and is the therapeutic agent in diabetes, and the other that either produces the so-called "hypoglycemic convulsions" or acts as an agent in producing the product, most likely one of protein decomposition. The author produced an insulin chloride by continued washing. This was less soluble in water, and had a smaller insulin titer. Convulsions were not observed following doses as high as 120 units, although the blood sugar was reduced to a minimum. The chemical properties and both the physiologic and therapeutic actions of this insulin will be reported in a later paper.

Stulik, Chicago.

Nervous Diseases

The Value and Safety of a Simplified Method of Pneumo-Encephalomyelography. Frank Liberson, Am. J. Roentgenol. **15**:231 (March) 1926.

By simultaneously replacing from 25 to 100 cc. of the cerebrospinal fluid with air, either in the ventricle through a trephine opening or in the spinal canal through lumbar puncture, the percentage of correct roentgenologic diagnoses of tumors was increased 35 per cent. Some of the immediate after effects cited were a sense of cranial succussion, while the ventricles were partly filled, headache, nausea, vomiting, pain and slight elevation of temperature. Collapse resembling fainting occurred in two of the forty patients examined. Because of its applicability and relative safety, it is hoped that this method may serve to broaden the usefulness of the roentgen ray in the diagnosis of cerebrospinal conditions other than tumors of the brain.

Stulik, Chicago.

Conduct Disorders of Children. Ira S. Wile, J. A. M. A. **88**:1022 (April 16) 1927.

Conduct disorders of children may be occasional, periodic or habitual, varying with time, place and circumstance as well as with age. The process of formation of character is a by-product of living, and conduct is one phase of the expression of its development. Conduct disorders are nonconformities of persons inadequately adjusted to their environment. The problem is to seek the cause of the difficulty. The conduct disorder must never be the object of treatment—one must treat the whole child in the total situation.

EPIDEMIC MENINGOCOCCUS MENINGITIS. JOHN P. CAFFEY, STAFFORD MCLEAN and RUTH C. SULLIVAN, J. A. M. A. **88**:1859 (June 11) 1927.

Increase in the sugar of the cerebrospinal fluid during specific serum therapy is a favorable prognostic sign. Before treatment in 90 per cent of the cases, the determination of sugar was low. In children more than 6 months of age, a failure of the sugar of the cerebrospinal fluid to increase during serum therapy is an unfavorable prognostic sign. When the infant is under 6 months of age, this is not necessarily the case. Quantitative determinations of the cerebrospinal fluid chlorides do not aid in the diagnosis or prognosis.

EPILEPSY IN CHILDHOOD. M. G. PETERMAN, J. A. M .A. **88**:1869 (June 11) 1927.

The author believes that the immediate cause of the epileptic convulsion is probably a disorder of the metabolism and may be a shift of the acid-base equilibrium toward the alkaline side. The treatment may be summarized in a few words: diet, phenobarbital (and its salts) and psychotherapy. The most striking and effective treatment is starvation, but this is only a temporary measure. The ketogenic diet, which is given following one week of starvation, consists of a ration low in carbohydrate and protein and light in fat adjusted for the individual patient. This produces a ketosis with a tendency toward acidosis, and its success rests with the ability to carry out the prescribed diet in careful detail. Later, the diet may be brought back to normal. Fourteen epileptic children have completed the diet and are now on normal diets. All have been free from attacks for from six months to three years.

THE TREATMENT OF EPILEPSY IN CHILDHOOD. HENRY F. HELMHOLZ, J. A. M. A. **88**:2078 (June 25) 1927.

Five years experience with the ketogenic diet for epilepsy has shown that 31 per cent of the patients will be free from attacks and that 23 per cent will be definitely improved. Some of the failures may be accounted for by inability to control the diet in the home. The diet is not easy to carry out, and it is difficult often to get the parents to continue it, when early results are not obtained.

BONAR, Salt Lake City.

HEREDITARY INFLUENCES IN POSTENCEPHALITIC PSYCHIC DISTURBANCES. U. FLECK, Arch. f. Psychiat. **79**:509 (Feb.) 1927.

The results of a study of hereditary data in fifty-two cases of metencephalitis showing psychic disturbances are presented. The main thesis followed by the author deals with the possibility of discovering whether the frequent occurrence of psychopathic trends in the family tree tends to determine the nature of the postencephalitic sequelae. In the fifty-two cases, thirty-five patients were below the age of 20. All the cases showed some mental deviations, twenty-seven of them representing the different types of psychopathies. Practically all of the psychopathies were found among the patients under 20 years of age. The study of the family trees of these persons included siblings of the patient and their parents and grandparents. Mental deviations noted were those of asocial tendencies, psychopathies, schizophrenias and manic-depressive psychoses.

The results can be summarized as follows: 1. Most postencephalitic patients below the age of 20 with psychopathic character changes showed a much greater frequency of psychopathic traits in the family tree than the older postencephalitic or normal individuals (the latter two being about equal). 2. This and other studies showed that psychopathic traits in the families of normal persons, as well as those who were subject to influenza but who had not developed encephalitis and also those who had developed encephalitis but had not shown any psychopathic manifestations following it, are about equally represented. Thus, heredity does not play any definite rôle in predisposing a

person to encephalitis or its sequelae other than psychopathies. 3. An analysis of the type and intensity of the psychopathic traits presented in this work would point to the fact that the frequent occurrence of psychopathic traits in the family is of a more pathoplastic than pathogenetic importance. It is the nature of the psychopathy rather than its occurrence that seems to be predetermined by hereditary influences.

MALAMUD, Foxborough, Mass.

PERIODIC DECREASE IN THE ABILITIES OF TALENTED CHILDREN. ITS CAUSE AND TREATMENT. HANS ECKERT, Gesundheit. f. d. Kindesalter **2**:213 (April) 1927.

It has been noted that children between the ages of 8 and 16 sometimes show a sudden decrease in their ability at school without any apparent psychic or physical reason. Eckert described these children as neurotic, with the typical symptom-complex of irritable weakness, lack of appetite, vasomotor lability and an excessive growth in height in contrast with the increase in weight. He also noted the presence of frequent pharyngitis which leads to a dry throat and an excessive intake of water, which in itself again aids the development of exudative phenomena on the mucous membranes. Therapeutically, he suggested the usual method of wisely ignoring the neurotic symptoms and the general physical training to develop the peripheral vasomotor centers and increase the child's resistance to changes of temperature.

THE DEAF AND DUMB CHILD. SCHORSCH, Gesundheit. f. d. Kindesalter **2**:238 (April) 1927.

The author points out that, before entering school, the deaf and dumb child is entirely neglected and valuable time is lost. He recommends the booklet of Marget Martin of Glasgow, "What the Mother of a Deaf Child Can Do," in which in simple words the parents are taught to start the child on the road of lip-reading. Schorsch is of the opinion that aside from this defect the deaf-mute is not under the standard of a normal child.

REICHLE, Cleveland.

THE SUGAR METABOLISM IN CHRONIC EPIDEMIC ENCEPHALITIS AND PARALYSIS AGITANS. R. A. TKATSCHEW and W. W. AXENOW, Ztschr. f. d. ges. Neurol. u. Psychiat. **104**:391 (Sept.) 1926.

Claude Bernard first demonstrated the fact that injury of the floor of the fourth ventricle causes hyperglycemia and glycosuria. More recently Brugsch, Dresel and Levy pointed out that the area punctured by Bernard corresponded to the dorsal nucleus of the vagus. Injury of the middle and caudal portion of this area caused hypoglycemia, while injury of the anterior or cephalic portion caused hyperglycemia. Brugsch, Dresel and Levy found two vegetative centers in the medulla: one, the sympathetic, which innervated the suprarenals, and the other, the parasympathetic, which innervated the pancreas. By studies in secondary degeneration they were able to demonstrate a center in the nucleus periventricularis which regulates the work of the medullary centers and maintains the sugar content in the blood. They demonstrated, moreover, a higher center localized in the pallidum, and were able to show changes therein in diabetes mellitus. Spatz and Lubarsch, however, consider the observations of these authors on the pallidum of diabetic persons as being wholly within normal limits.

Although Brugsch, Dresel and Levy defend their views with few facts, they at least offer a new point of view. Experimental and clinical work recently has shown that important vegetative centers lie in the midbrain; centers for albumin, fat, and water metabolism and for temperature lie there. Moreover, the histologic structure of the cell groups in the midbrain — nuclei supraopticus, paramedianus, paraventricularis, substantia grisea — is similar to that of the sympathetic cells.

Tkatschew and Axenow studied the sugar metabolism in cases of chronic encephalitis and in paralysis agitans on the basis of the work of Brugsch, Dresel and Levy. Their patients were assumed to have disease of the basal ganglia and midbrain. Fasting sugar contents of the blood and urine were determined in their cases, from 50 to 100 Gm. of dextrose were given, and the sugar content of blood and sugar studied for the next five hours. The microchemical method of Bang was used in the determinations. Thirteen cases of postencephalitic parkinsonism, six cases of morbus parkinsoni, and one case of double athetosis were studied. According to the authors' interpretation, all thirteen cases of postencephalitic parkinsonism showed disturbance in the sugar metabolism. In plotting the sugar curves, a return to normal was not found after five hours in one case, a return to normal was found during the fifth hour in two cases and during the third hour in five cases. In one case the blood sugar rose to 255. In four of six cases of Parkinson's disease a disturbance was found. Return to the normal level occurred in the fourth hour in the case of double athetosis. In their cases of postencephalitic parkinsonism they demonstrate a marked disturbance in eight instances, slight in three cases, and normal in two.

ALPERS, Philadelphia.

VACCINATION DISTURBANCES OF THE CENTRAL NERVOUS SYSTEM. M. GIOSEFFI, Ztschr. f. Kinderh. **43**:321 (March) 1927.

A breast-fed infant, aged 8 months, was seized with a convulsion and subsequent fever five days after vaccination against smallpox. Another convulsion occurred the next night and one again three days later. The infant was drowsy during the day, refusing to nurse, but she was restless at night. In three weeks, after a loss of 1,200 Gm., she began to mend.

EMERSON, Boston.

TWO CASES OF HYPOPLASIA PONTONEOCEREBELLARIS. S. KOSTER, Acta psychiat. et neurol. **1**:47, 1926.

The author presents a pathologic study of two cases of hypoplasia of the pons and neocerebellum. His first case was that of a girl, who died from diphtheria at the age of 1 year. Further clinical data were not available. The posterior fossa and its contents were very small. The cerebellum and the brain stem together weighed only 15 Gm. The cerebellar hemispheres appeared as two flattened disks, and this poor development left the anterior part of the vermis uncovered. Only a few primary convolutions were present, except in the lateral and ventral parts, where there were a few secondary lobuli, and dorsally and caudally, where a few secondary and tertiary lobuli could be distinguished. The parts that showed a greater degree of development corresponded to the lobus cuneiformis. The vermis, flocculi and tonsils, though small, were normal. Only a small portion of the pons was present. The pyramidal and olivary protuberances were of normal size.

Histologically, the stratification of the cerebellar cortex was normal. The molecular and granular zones of the hemispheres were thin, and in the areas where this thinning occurred the number of Purkinje cells was less than normal. In the lateral part of the lobus anterior and lobus simplex, the Purkinje cells were small and slender. The central white matter was so poorly developed that the nucleus dentatus was almost in touch with the gray matter of the cortex. The radiation of the middle cerebellar peduncles could scarcely be distinguished. The white matter of the vermis was normal. The convoluted form of the dentate nucleus had disappeared and the nucleus consisted of only small nests of cells surrounded by myelinized fibers. These cells were present only in the dorsomedial (paleocerebellar) part. The nucleus emboliformis and nucleus tecti were more nearly normal than was the dentate nucleus, but they were small. The nucleus globosus was normal. The superior and inferior cerebellar peduncles were diminished greatly in size, while the middle consisted only of a few fiber bundles.

The nuclei arcuati were small, and the cells were of embryologic type. Only a few cells of the corpus pontobulbare could be distinguished. With the exception of the pyramidal bundles, the basis pontis was nearly absent. The frontopontile fibers were normal as far as the region of the root fibers of the facial nerve, caudad to which they could not be found. The temporopontile tract was normal as far as the root fibers of the trigeminal nerve. A few fibers in the basis pontis represented the strata superficiale, profundum and complexum. Most of the cells of the pontile nuclei were absent, more so in the caudal than in the cephalad parts. The inferior olivary nuclei were small. The lateral portions of the olivary convolutions showed the most abnormalities, and, except at the caudal pole, the ventral portions were developed best. In the portions of the nucleus in which no cells were found, the tissue appeared undifferentiated. Only a thin olivary capsule was present. The caudal part of the red nucleus was small, and only large cells were found. The capsule was absent. The caudate nucleus was very large.

The second case was that of a child, aged 21 months, whose clinical history was unknown. A condition almost identical with that in the first case was found on pathologic examination, except that there was a heterotopia of Purkinje cells in one cerebellar tonsil, and the inferior olivary nuclei were better developed.

In discussing these cases the author points out that the pons, nuclei arcuati and inferior olivary nuclei develop from cells situated in the medulla, close to the lateral border of the fourth ventricle. From this area, the cells migrate in two directions, presumably under neurobiotactic influences. One group passes between the fibers of the seventh and eighth cranial nerves, lateral to the restiform body, in the direction of the frontoparietotemporopontile tracts, to form the cells of the pons. The other group migrates in a ventrocaudal direction to form the inferior olivary nucleus and the arcuate nuclei. The cerebellum develops from a germinal center in the roof of the fourth ventricle contiguous to the area just described. Both Winkler and Brouwer consider that if a pathologic factor operates on these adjacent areas before the migration of the cells has begun the result will be a hypoplasia of the basis pontis, cerebellum and inferior olivary nuclei. The migration takes place earliest to the last mentioned nuclei, that to the pons occurring latest. In the author's cases the etiologic process must have taken effect after the migration to the olives was nearly complete but before that to the basis pontis, cerebellum and arcuate nuclei had commenced. This would be about the third fetal month.

Besides the injury to the germinal centers, he considers that that part of the hypoplasia, particularly that affecting the inferior olives, is the result of what he calls the correlative factor, i. e., an injury to one part of the developing nervous system which produced maldevelopment of those parts connected with it. In the instance of the hypoplasia of the basis pontis, Koster thinks the influence of the correlative factor has a bearing also, for the consensus of opinion is that the pontile nuclei send their axons mostly to the contralateral cerebellar hemisphere, although a few may go to the ipsilateral one, but none go to other parts of the brain.

The location of the hypoplasia follows the law that developmental defects implicate the newer structures most. From numerous phylogenetic and ontogenetic studies, the cerebellum has been divided into two portions, the paleocerebellum — the vermis, flocculus and paraflocculus — and the neocerebellum — the cerebellar hemispheres. Van Valkenburg considered that the lateral parts of the lobus anterior and a small part of the lobus simplex formed a transition zone between the new and the old structures. In Koster's cases the neocerebellum was hypoplastic, and the defects included these areas. It seems reasonable to conclude that they are really parts of the neocerebellum. In regard to the cerebellar nuclei, the ventrolateral-caudal portion of the dentate nucleus is a new structure; the remainder of this nucleus and the other nuclei are old. The former part shows marked hypoplasia. Also the hypoplasia of the inferior

olivary nuclei follows the phylogenetic and ontogenetic development of these structures. The best preserved parts are the oldest, i. e., the medioventral and dorsal accessory olives. Brouwer is of the opinion that the oral part of the main nucleus was old also, but in Koster's cases this was hypoplastic. It is well known that the inferior olive is projected onto the cerebellar hemispheres, and the results of the studies of other authors indicate that the oral pole is projected onto the lateral portions of the anterior lobes. As has been mentioned already, Koster considers these portions to be new structures which would account for the defect in the oral pole of the olives. Furthermore, a restudy of the case on which Brouwer formulated his opinion shows that the lateral part of the anterior lobes was degenerated, which would account for the hypoplasia of the oral part only of the olives.

Koster's cases substantiate the theory of Masuda that the frontal part of the pons is connected with the caudal part of the cerebellum. Also the fact that there is no connection between the frontopontile and temporopontile tracts and the caudal part of the pons confirms Kapper's opinion that the latter is the oldest portion phylogenetically.

PEARSON, Philadelphia.

HISTOLOGIC OBSERVATIONS IN TWO CASES OF JUVENILE PROGRESSIVE PARALYSIS. MARIE SPRINGLOVA, Čas. lék. česk. **64**:1833 and 1880 (Dec.) 1925.

Histologic observations of the nervous tissues are given in minute detail in the case of a girl, aged 13 and a boy, aged 16; the latter also had atrophy of the testis and thymus. Purkinje cells, long regarded as specific in juvenile progressive paralysis, have been demonstrated by various workers in senile dementia, tuberculous meningitis, delirium tremens and dementia praecox. The juvenile type is differentiated from the adult type in that it forms on the basis of an underlying congenital syphilis. It is uncertain whether the syphilitic toxins produce changes in the central nervous system during fetal life. That the disease is purely endogenous cannot be proved to date. In the juvenile type, atrophy of the affected nervous tissue is relatively greater, the intensity of the process possibly depending on the age. The double and polynuclear Purkinje cells with dendritic and neuritic changes are more likely to be found in the juvenile type.

STULIK, Chicago.

Diseases of the Ear, Nose and Throat

AN APPARATUS FOR THE STEREOSCOPIC ROENTGENOGRAPHY OF THE MASTOIDS. JAMES B. BULLITT, Am. J. Roentgenol. **15**:256 (March) 1926.

If the patient is placed on his back with the head within a double plate-holder box with lateral circular openings, both mastoids can be visualized by simply moving the tube. In this position pressure is not produced on the tender mastoid, and both mastoids are in full view for accurate placing.

STULIK, Chicago.

ACUTE OTITIS IN INFANTS. L. W. DEAN, Arch. Otolaryng. **6**:201 (Sept.) 1927.

A review of the literature is given from 1894 to the present. From his observations, the author concludes that otitis in infants may cause refusal of food, malnutrition with loss of weight, diarrhea, excoriation of the buttocks, anhydremia, the syndrome of cholera infantum and possibly syncopal attacks. His treatment depends more on these symptoms than on purely otologic ones. Otitis can always be diagnosed if present. Disease of the paranasal sinuses may cause all the same symptoms as otitis except syncopal attacks.

Much emphasis is laid on the necessity of complete confidence and cooperation between the otolaryngologist and the pediatrician in these cases.

BARNETT, Chicago.

NASAL ACCESSORY SINUSITIS IN CHILDREN. ALBERT H. ALAND, California & West. Med. **27**:74, 1927.

Sinusitis is frequently overlooked in children because of the misconception still prevalent that sinuses in children are undeveloped and therefore not foci of infection. Many prominent rhinologists and internists believe that the relation of focal lesions in the accessory nasal cavities of children to systemic disease, such as arthritis, endocarditis and nephritis, is of equal importance to that of tonsillar and dental disease. Numerous patients with systemic infection, orbital abscesses, optic neuritis and especially infectious arthritis, who are from 6 months to 14 years of age, are reported cured by treatment of infection of the sinuses.

The author's youngest patient was 7 months of age with a history of an orbital abscess of the right eye and osteomyelitis of the fourth metacarpal bone of the left hand at 1 month of age. Both were opened and drained at the same time. When the infant was 3 months old, an orthepedic surgeon curetted the metacarpal abscess and applied an extension splint because of a half inch shortening of the bone. When the infant was aged 7 months, as the ethmoid suppuration was persistent and the sinus over the metacarpal bone did not heal, the author did an external operation on the right anterior ethmoid cells, which were found full of pus and granulations. Thirteen days later, the wound was dry and healed and the sinus over the metacarpal bone of six months' duration also healed.

There was no recurrence of infection when the patient was seen one year later.

STREPTOCOCCUS MUCUS INFECTION CAUSING LATERAL SINUS THROMBOSIS. H. J. PROFANT, California & West. Med. **27**:364, 1927.

The author lays particular emphasis on observing what may seem even a mild infection of the middle ear.

A boy, aged 7, had mild acute rhinitis. Two days later, he complained of pain in the right ear. Examination showed slight injection of the drum but no bulging. The temperature was 100. After four days, pain subsided. Three days later, he went to school and was apparently well for ten days. He then became suddenly ill, with a temperature of 104 and signs of beginning meningitis; but he did not complain of pain in the ear. On the second day the symptoms of meningitis were less marked, but the temperature was still high.

On the third day, the temperature was lower, and the symptoms of meningitis disappeared. Later, there was a severe chill for fifteen minutes and a temperature of 105, followed by profuse sweat. The chill and fever recurred in twenty-four hours and then at shorter intervals. There was no nystagmus, and the fundi were normal; signs of meningitis were not present; the nose and throat were normal; the right mastoid was not tender. The ear drum was dull, with no light reflex, and appeared thick. Paracentesis did not show pus, but there was a peculiar thick feeling on incision, which Ruttin has described as a feeling of cutting bacon. There was no redness in the canal and no sagging of the posterior-superior wall. Immediate exploration of the mastoid was advised. When the cortex was opened, superficial cells appeared normal. When the antrum was entered through dense bone, no pus but a small amount of thick grayish red granulation tissue was found. When the incision was continued posteriorly, an abscess at the knee of the sinus was suddenly entered. The dura and sinus had been exposed by the infection, and the abscess localized at this point. The operation was stopped on account of the patient's condition. The next morning he was much better, but twenty-four hours later had a cycle of chills and fever and then another in ten hours. Under a light anesthetic, the sinus was exposed and an obturating thrombus was found. The surgeon's associates quickly ligated the internal jugular above the facial vein; the thrombus was removed, and free bleeding was obtained above and below. There were no more chills and fever, but the boy

was septic. Ten cubic centimeters of 1 per cent mercurochrome-220 soluble was given intravenously on the first and third days following the operation, and a slow recovery followed.

The most valuable lesson from this case is summed up in the adage: "Be concerned about every ear infection, no matter how mild it may seem."

Other important conclusions are:

1. *Streptococcus mucosus* is a treacherous infection of the ear because of the mild onset, the long latent period and the tendency to attack bone.

2. *Streptococcus mucosus* is classified by some bacteriologists as a pneumococcus mucosus.

3. A two-stage operation proved successful: first, the mastoidectomy which established drainage; second, the ligation of the internal jugular vein and removal of the infected thrombus.

4. Intravenous injection of 1 per cent mercurochrome-220 soluble aided in combating the sepsis.

JEIDELL, Salt Lake City.

PARACENTESIS TYMPANI: A PRACTITIONER'S OPERATION. W. STUART-LOW, Practitioner **119**:57 (July) 1927.

It is deplorable to find that so many infants and children in hospital clinics and in private practice have discharging ears. It is the author's opinion that this is largely the result of an initial infection of the tympanic cavity not being properly diagnosed and treated.

The membrane is always red; sometimes it is intensely so in acute otitis media and is bulging as well. It is not necessary for pain to be present, although often intense throbbing is felt accompanied or not by fever.

It is the author's opinion that paracentesis tympani should be performed at once and that this should be done under chloroform anesthesia in all cases.

Since nearly all tympanic infections are secondary to nasopharyngeal sepsis, the chief prophylactic measure consists in the thorough removal of all tonsils and adenoids.

The emphasis in the article is on training the general practitioner in the performance of this operation.

GELSTON, San Francisco.

INJECTIONS OF LIPIODOL TRANSGLOTTICALLY IN CHILDREN. C. J. A. VAN ITERSON and D. J. STERNTUIS, Arch. internat. de laryngol. otol. rhinol. **6**:399, 1927.

The various methods of injecting iodized oil 40 per cent into the trachea and lungs were reviewed. The authors use the same method and instruments in children as in adults. The pharynx is rendered insensible with a 1 per cent solution of cocaine. The larynx is then anesthetized with a 10 per cent solution of cocaine. The oil, slightly tepid, is introduced by the indirect method by means of a syringe with a curved cannula. The cannula has several openings, but the end is closed. From 20 to 30 cc. is usually sufficient in children.

ROTH, Kankakee.

EARLY TONSILLECTOMY IN PERITONSILLAR ABSCESS. W. HAARDT, Wien. klin. Wchnschr. **40**:1173 (Sept. 15) 1927.

Following recent reports of the advantages of treating peritonsillar abscess by immediate removal of the tonsils in place of simple incision, Haardt determined to try a modification of this method. In twenty-one patients (presumably adults), he first incised the abscess. From three to seven days later, when there was no further drainage of pus and the fever and local signs of inflammation had subsided, he performed tonsillectomy. He found the operation technically easy at this time and was not troubled by postoperative hemorrhage. Local anesthesia was used in seventeen cases with good results.

All of the patients did well following the operation except two, who developed postoperative sepsis. One of these made a recovery without complications. The other had pyemia, endocarditis and empyema necessitating resection of the ribs, but he too survived.

Haardt is convinced that, although there is a risk in allowing an incompletely evacuated peritonsillar abscess to exist, it is likewise dangerous to perform tonsillectomy while acute local inflammation is present. He recommends that the tonsils not be removed until the abscess has completely healed. The only exceptions to this rule are persistent bleeding or incomplete drainage from a peritonsillar abscess, or when a general sepsis is already present.

OLMSTED, Seattle.

CRITICAL REMARKS ON MENINGITIS SEROSA. OTTO FLEISCHMANN, Ztschr. f. Hals-, Nasen- u. Ohrenh. **17**:547 (March 18) 1927.

Kobrak thinks that the name "otogenous acute hydrocephalus" should replace the term "serous meningitis." Contrary to the condition of other serous cavities —the pericardium, the pleural and peritoneal cavities—the meninges offer further pathologic possibilities, namely, a disturbance of the normal production and resorption of the cerebrospinal fluid. A pathologic increase of the fluid can occur without signs of inflammation, as, for instance, in anemia, chlorosis and similar conditions. The author has stated repeatedly that extradural foci of pus, for instance, those of the middle ear cavities or nasal accessory sinuses, could cause an increase of the cerebrospinal fluid by irritation without inflammatory processes in the meninges. An increased production of the spinal fluid is found in tumors and abscesses of the brain and in cases of circumscribed meningitis. The eye shows similar conditions. The intra-ocular fluid can be increased independent of inflammatory conditions of the eye. In a spinal fluid which shows no changes on repeated examination, especially if there is no increase in albumin, meningitis is excluded. If a complete spinal block occurs, without symptoms of inflammatory changes, the fluid regularly found in the distant part must be caused by transudative and exudative processes, respectively. The high albumin content and small number of cells characteristic of the fluid below the place of interruption indicate a serous-exudative inflammatory process and prove that serous meningitis does not act differently from inflammation in other serous surfaces. Preysing, Holzmann, Zange, Pappenheim and others reserved the term "meningitis serosa" for forms in which an increase of albumin and of the number of cells is present, whereas forms with increase of fluid alone are named "liquor acutus," "congestion of fluid" or "hydrops cerebrospinalis." Bonhoeffer stated that there is no clear anatomic view concerning meningitis serosa and that no difference exists anatomically, clinically or pathogenetically between it and other acute and chronic cases of hydrocephalus. The author does not deny the existence of a serous meningitis, yet neither the clinical picture nor the spinal fluid differ greatly from the lighter forms of purulent meningitis, especially the circumscribed form. The author differentiates between simple meningism, acute inflammatory hydrocephalus, collateral meningitis and diffuse suppurative meningitis. It is disturbing to find that the fluid obtained by spinal puncture shows only the change of the meninges in the region in which it was procured, so that, at times, little change is found, even if the brain is covered with pus.

THE GRAPHIC METHOD OF TEACHING DEAF-MUTES. E. W. SCRIPTURE, Ztschr. f. Hals-, Nasen- u. Ohrenh. **19**:27 (May 16) 1927.

When a totally deaf child, who has never learned to speak, enters school in order to learn how to speak, its organs of voice production are normal, as a rule, and it would be able to use its voice like other people if it would know how. The speech of such a child is frequently faulty. The graphic method, first worked out by Abbé Rousselot, can be used to correct many

mistakes of the pupil. The author published his first results in the *Volta Review* in 1913. The person talks into a mouthpiece. The motion of the air and of the sound waves is carried through a wide tube to a membrane made of oil silk. The movements of the membrane are magnified by a lever and registered on the surface, which is covered with soot. First the child speaks into the apparatus, and then the teacher does so for comparison. The method should be used from the start, so that the child will learn the correct enunciation. It is much better to prevent a faulty enunciation than to be obliged to correct it later. According to the experiences of Freunthaller, it is important to begin the teaching of speech at the age of 3 years.

AMBERG, Detroit.

EXPERIMENTAL INVESTIGATION OF THE TONSILLAR FUNCTION. VIGGO SCHMIDT, Ugeskr. f. Laeger **89**:737, 1927.

Massage of healthy tonsils of young persons has been shown to produce a marked but temporary leukopenia. Subcutaneous injection of a saline extract, prepared from tonsillar tissue with functioning germinal centers, shows a similar lowering of the leukocytes. Extracts from organs other than lymph glands with germinal centers would not produce this effect. The author believed that healthy tonsils of young persons secrete a substance which inhibits leukocytosis.

SCHLUTZ, Minneapolis.

Diseases of the Skin

THE RELIEF OF ITCHING BY EPINEPHRINE IN CERTAIN CASES OF INFANTILE ECZEMA. T. D. PILCHER, J. A. M. A. **89**:110 (July 9) 1927.

Subcutaneous or intramuscular injections of epinephrine hydrochloride in doses of from 0.1 to 0.3 cc. of 1:1,000 solution appear to give temporary relief from the severe discomfort of certain eczematous infants as it does in urticaria. Other than the sedative action lessening the effect of the trauma from scratching, there is insufficient data to warrant an opinion as to whether epinephrine is otherwise beneficial in eczema.

BONAR, Salt Lake City.

RINGWORM OF THE SCALP TREATED BY THALLIUM ACETATE EPILATION. G. B. DOWLING, Proc. Roy. Soc. Med. **20**:71 (May) 1927.

The technic advised by Buschke was followed; that is, a single dose of thallium acetate in sweetened water was given by mouth. The actual dose given in one case was 8 mg. per kilogram of body weight; in the other two, 8.5 mg. Four cases were presented. The patient should be carefully weighed, preferably twice on different days. The dose of thallium acetate must be accurately calculated to the nearest milligram and administered in water. Buschke found that mixing the thallium with food was unsatisfactory, as the epilation partly failed. The hair begins to loosen on the seventh day, falls out rapidly between the fourteenth and nineteenth day, and is all out by the nineteenth day. Regrowth begins practically at once. A curious occasional feature in the early stage of regrowth is the presence of enormous numbers of exclamation-mark hairs scattered all over the scalp. The infected hairs seem to resist epilation more readily than do the normal hairs, but they usually fall a few days later. Toxic reactions were mild, and the patient was relieved by rest in bed. In practically every case the patients complained of pains in the lower limbs, with some loss of appetite in a few cases, and in one case headache was a transient symptom occurring at the time of epilation. The pains in the limbs occur about ten days after the dose, and pass off at about the time of epilation, from eight to ten days later. No symptoms of any kind have been observed in the first week. In Buschke's cases there were no toxic symptoms of any kind, and he was able to produce complete epilation with

8 mg. per kilogram of body weight. Therefore the conclusions are that: 1. The thalium used should be biologically standardized. 2. It should be freshly made and absolutely pure. 3. The patients should, if possible, be kept in bed throughout the treatment.

GUY, Pittsburgh.

ULCERATED ANGIOMA OF THE FACE. MILIAN and DELARUE, Bull. Soc. franç. de dermat. et syph. **34**:189, 1927.

In an infant, aged 9 months, the rupture of a cavernous angioma apparently was spontaneous. The ulceration was extensive and deep.

PARKHURST, Toledo, Ohio.

ZOSTER AND VARICELLA BY BIOTROPISM. LOTTE, Bull. Soc. franç de dermat. et syph. **34**:237, 1927.

A child with pertussis had received injections of serum. Eleven days after the first injection, and following a mild serum reaction of two days' duration, zoster and varicella developed simultaneously. The patient had been exposed to varicella two months previously.

REMARKABLE ACTION OF BISMUTH IN TWO CASES OF LUPUS ERYTHEMATOSUS. A. SEZARY and F. BENOIST, Bull. Soc. franç. de dermat. et syph. **34**:382, 1927.

A case of lupus erythematosus of five months' duration cleared up after six weeks of treatment with bismuth, comprising twelve injections. A second patient, whose condition was of four months' standing, was greatly improved after ten injections.

PROBABLE LICHEN PLANUS ATROPHICUS OF ZOSTERIFORM DISTRIBUTION IN A CHILD AGED THREE YEARS. HUDELLO, CAILLIAU and MORNET, Bull. Soc. franç de dermat. et syph. **34**:385, 1927.

The eruption, of eleven months' duration, involved the right side extending from the buttocks to the toes, and was not absolutely typical of lichen planus atrophicus. The histologic observations are described.

GLOSSITIS WITH LEUKOPLASTIC PATCHES, AND FACIAL DERMATITIS OF BACTERIAL ORIGIN; MORBID PARALLELISM. HUDELLO, MORNET and CAILLIAU, Bull. Soc. franç. de dermat. et syph. **34**:387, 1927.

The clinical pictures of the condition which occurred in two brothers with congenital syphilis were strikingly similar, and stigmas of syphilis were present. The lingual patches were possibly due to a local infection, and may not have been true leukoplakia.

HYPERKERATOTIC PSORIASIS WITH PAPILLOMATOSIS AND CUTANEOUS HORNS; LINGUAL LESIONS OF UNDETERMINED NATURE. HUDELO, GARNIER and CAILLIAU, Bull. Soc. franç. de dermat. et syph. **34**:389, 1927.

The psoriasis was of twenty-five years' duration, in a woman, aged 37. The histologic observations in the cutaneous and lingual lesions are described.

EDEMATOUS VESICULAR ERYTHRODERMA CURED BY THE EXCISION OF INTRAGLUTEAL NODULES OF BISMUTH. MILIAN and LENORMAND, Bull. Soc. franç. de dermat. et syph. **34**:395, 1927.

Nodules of bismuth were excised from both buttocks, and the erythroderma, which had been present for eight months, promptly faded.

LARGE ANGIOMA AND TUMOR OF THE ARM IN A NEW-BORN INFANT. LOTTE, Bull. Soc. franç. de dermat. et syph. **34**:402, 1927.

An orange-sized hemangioma partially encircled the arm.

CENTRIFUGAL ANNULAR ERYTHEMA. JEANSELME and BURNIER, Bull. Soc. franç. de dermat. et syph. **34**:413, 1927.

The authors describe a case in a boy, aged 14, with generalized involvement of one year's duration. Photographs are included. The etiology was obscure. The literature on the subject is briefly reviewed. Lipschutz first described the condition and called it erythema chronicum migrans. In some cases, bites from insects appear to be causative. ARCH. DERMAT. & SYPH.

Surgery and Orthopedics

TRAUMATIC COXA VARA: A CASE REPORT. WILLIAM COLIN, Am. J. Roentgenol **15**:242 (March) 1926.

In a boy, aged 17, with traumatic coxa vara, the condition was erroneously diagnosed as a fracture through the neck of the femur with shortening of the neck, because of the epiphyseal line in the roentgenogram. Epiphyseal displacement must also be considered in injuries about the hip in adolescence.

CONGENITAL DISLOCATION OF THE HIP. A METHOD OF DETERMINING THE DEGREE OF ANTETORSION OF THE FEMORAL NECK. STEELE E. STEWART and ROLLA G. KARSHNER, Am. J. Roentgenol. **15**:258 (March) 1926.

The patient is placed prone on the abdomen and the knees are flexed 90 degrees so that the legs are vertical. With a goniometer needle attached to the leg, the latter is moved across the midline of the body, producing outward rotation of the femur, the arc of which is measured by the goniometer. The fluoroscopic check shows that the angle between the neck and the shaft of the femur increases until it becomes a straight line. If the rotation is continued beyond this, the neck will deviate outward. At this point the dial reading is taken and the process repeated for checking. This maneuver is applicable only to dislocated hips, the normal hip allowing only limited rotation of the femur. In a femur in which the neck joins the shaft without any anterior deflection, 90 degrees rotation is required to place the neck in a sagittal plane. Therefore, rotation through less than 90 degrees, which would bring the neck and shaft into a straight line, would vary from 90 degrees by the amount of anterotation already existing in the femoral neck. STULIK, Chicago.

RHABDO-MYOSARCOMA OF THE VULVA IN CHILDREN. C. D. LOCKWOOD, Arch. Surg. **14**:860, 1927.

There are forty-one cases of this tumor on record. The case described was in a child, aged 16 months. The tumor was first diagnosed endothelioma or spindle cell sarcoma, and treated with radium. In nine months abdominal metastases occurred. At postmortem, tumors were found in the abdomen, pericardium and left lung. The structure was rather myxomatous with large spindle cells containing cross and longitudinal striations.

THE TREATMENT OF PATIENTS WITH FUNNEL CHEST. J. ZAHRADNICEK, Čas. lék. česk. **64**:1814 (Dec.) 1925.

Funnel chest may be congenital, due to pressure of the elbow, knee or chin associated with micrognathia. As an acquired defect it is due to rickets, osteomalacia and trauma. In a boy, aged 16, with typical funnel chest associated with severe palpitation and dyspnea on the least exertion, the defect

was corrected by placing two bronze aluminum wires about the sternum through a small incision in the skin. With pulleys, a weight of from 1 to 2 Kg. was kept on these wires over a period of weeks with the patient in the prone position. The author thought that two periods of six weeks with an intervening rest period of one month should cure most patients. On reexamination three months after the patient's discharge with complete obliteration, it was found that 30 per cent of the deformity had recurred, but the symptoms were not present.

STULIK, Chicago.

Miscellaneous

EXTERNAL HEAT A CAUSE OF FEVER IN CHILDREN. KATHARINE DODD and SCOTT J. WILKINSON, J. A. M. A. **88**:787 (March 12) 1927.

It has been noted that external heat is usually not mentioned as a cause of obscure fever in children. Five infants were shown to have fever, irritability and restlessness caused by external heat, all of which were relieved by placing the children in comparatively cool rooms with moving air. Colored children are not similarly affected by the heat. During the summer months, extreme degrees of external temperature must be thought of as the cause of fever in white infants.

THE VITAL CAPACITY OF THE NEGRO CHILD. FRANK L. ROBERTS and JAMES A. CRABTREE, J. A. M. A. **88**:1951 (June 18) 1927.

The vital capacity of the negro children is lower than that of white children of the same age, the same body weight, and the same standing body height, but is practically the same as that of white children of the same stem length.

GROWTH OF CHILDREN THAT ARE BELOW AVERAGE WEIGHT. F. L. ROBERTS, J. A. M. A. **89**:847 (Sept. 10) 1927.

A study of the gain and loss of weight of 1,491 native born American children was made at a time when active nutrition work was not being carried on. The children were grouped into three classes: (1) those above or less than 7 per cent; (2) those from 7 to 12 per cent below the average weight and (3) those more than 12 per cent below the average. It was concluded that children above or equal to the average weight tend to remain in that group, while those below the average tend to remain below the average. The greater the underweight the greater the tendency to remain below the average. Girls are more prone to drop into lower groups and remain there. Children below the average weight gain as many pounds per inch increase in height as do children of average weight. Girls tend to lose weight more than boys Girls below the average weight do not gain as regularly as boys.

TRANSFUSION IN INFANCY AND CHILDHOOD. J. BUREN SIDBURY, J. A. M. A. **89**: 855 (Sept. 10) 1927.

A review of 556 cases of transfusion in the last ten years led the author to conclude that transfusion in infancy and childhood is a valuable therapeutic remedy which should be used more generally. It is essential that cross-matching of fresh specimens of blood be done before each transfusion, regardless of the age of the patient. Exsanguination transfusion is indicated in those patients given incompatible blood, and in the severe toxemias from burns, erysipelas, acute intestinal intoxication, toxic pneumonia, septicemia, infectious diarrhea and carbon monoxide poisoning. Protracted respiratory infections are greatly benefited and the course is shortened by one or more transfusions. Malnutrition with secondary anemia is benefited by transfusion without any dietary change. Too little emphasis is placed on the hemolysis reaction in blood matching. Five hundred and thirty-seven transfusions were given by the

Unger method, ten by the citrate method in the vein, five by the citrate method into the peritoneal cavity and four by the Lindemann method, two of which were given by the superior longitudinal sinus, and two through the umbilical vein. Only 2 per cent of the patients in whom transfusion was performed had chills.

BONAR, Salt Lake City.

EFFECT OF EPINEPHRINE ON THE BLOOD PICTURE IN NORMAL, LYMPHATIC AND SCROFULOUS CHILDREN. G. CAREDDU, Riv. di clin. pediat. **25**:543, 1927.

Epinephrine was injected intramuscularly, and the total and differential white blood cell count was made before, one-half hour, 1 hour and one and one-half hours following the injection. Three types of reaction were noticed: (1) mononuclear (relative increase of lymphocytes), (2) biphasic (relative increase of lymphocytes, followed by one of polynuclear cells) and (3) polynuclear (relative increase in polynuclear cells).

Three types of children were studied: (1) ten normal children; (2) ten tuberculous children, and (3) twelve so-called lymphatic children, who showed lymphatic hyperplasia but negative tuberculin reactions. The last group was composed of children having seborrhea, milk crusts, geographic tongue, enlarged thymus and adenoids.

The results obtained are given in the accompanying table.

	Polynuclear Per Cent	Biphasic Per Cent	Mononuclear Per Cent
Normal	50	20	30
Scrofulous	40	50	10
Lymphatic	17	58	25

Although "lymphatic" children show a tendency to have a mononuclear or biphasic reaction, this test with epinephrine is not considered to be of practical diagnostic importance.

HIGGINS, Boston.

ADDRESS AT THE PRUSSIAN CONFERENCE FOR SOCIAL SERVICE AMONG INFANTS AND SMALL CHILDREN, 1926. SCHLOSSMAN, Gesundheit. f. d. Kindesalter **2**:1 (Nov.) 1926.

The address represented a vindication of the use of homes for infants and small children in contrast to the foster mother plan adapted by Pfaundler. Schlossman believed that the increased mortality and hospitalism are preventable evils of such institutions. He called attention to his work in Düsseldorf since 1919. Under his regimen, the mortality among illegitimate children in certain groups reached lower levels than among the legitimate children.

INSTRUCTION FOR YOUNG GIRLS IN THE CARE OF INFANTS. E. LIEFMANN, Gesundheit. f. d. Kindesalter **2**:79 (Nov.) 1926.

THE LAWS PERTAINING TO EDUCATION OF TEACHERS FOR INFANT CARE AND THEIR VALUE. EDITH PAESLACK, ibid. **2**:86 (Nov.) 1926.

Both articles treat of the difficulties in obtaining trained teachers for the education of the young girl in the public schools of Germany in the proper care of infants. Liefmann was in favor of having the social service nurse take over this instruction. Paeslack wished to have the instruction carried out by the school teacher, as she believed that any other system would disturb the evolution of the centralized teaching which is now the aim of German public schools. She also believed that pedagogic training is as necessary as a special training in infant welfare.

THE PROBLEM OF EARLY INFANTILE MORTALITY. F. WENDENBURG, Gesundheit. f. d. Kindesalter **2**:107 (Jan.) 1927.

The article is concerned with the alarming increase in the mortality of infants in the first ten days or six weeks in contrast with the drop in the annual mortality. Among illegitimate infants, this figure in 1927 comprised 67 per cent of the entire annual infantile mortality, whereas the mortality of infants aged 6 weeks was only 41 per cent for 1913. The figures were obtained from a group of about 50,000 children in the industrial territory of the Ruhr. Wendenburg suggested that: 1. All general social service workers are to have a special training in infant social service. 2. The midwife, who plays such an important rôle in German obstetrics, should be utilized more by the social service bureau, in that she should be given some training in infant care, be held responsible for the reporting of all children below normal weight and length as well as all premature infants and that she be allowed to make use of some of the social service privileges for her clientele. 3. The premium paid needy mothers should be paid out in part before the birth, as in this way, it will be possible to promote the infant's development at the earliest state. The payment of the premium should be highly individual. 4. Births should be reported to the respective bureaus immediately, if necessary, by telephone each day. This is particularly necessary in country districts. 5. The statistics concerning the causes of infantile mortality in the first ten days of life should be fully itemized. Wendenburg realized the amount of work involved and suggested that sections of each county be chosen and that the statistics in these counties be gathered by a special social service worker.

OTTO HEUBNER'S WORK IN SOCIAL SERVICE. LEO LANGSTEIN, Gesundheit. f. d. Kindesalter **2**:204 (April) 1927.

Langstein gives a review of Heubner's initiative, intense interest and individual care in his career as social service hygienist.

HYPERTHERMIA IN THE INFANT DUE TO CRYING. H. RIETSCHEL-WÜRZBURG, Gesundheit. f. d. Kindesalter **2**:208 (April) 1927.

Rietschel is of the opinion that hyperthermia in an infant is possible when excessive crying plus too close bundling and too low a fluid intake prevails.

REICHLE, Cleveland.

THE CHILD SOCIETY FEARS. MAURICE ROUVROY, Revue internat. de l'enf. **3**:333, 1927.

One reads much of the child who brings joy and comfort and of the child who arouses pity and concern, but few consider the one who brings fear and dismay. People are afraid of the unusual and the abnormal as well as the potential criminal, and confine them as promptly as possible in institutions. In an examination of 4,580 such cases, the author has analyzed the causes and classified the types of lawlessness. In 2,855 instances, the fault could be traced directly to bad environment during the most impressionable period of life. Reconstruction of this group in every country is a crying need of the hour. Rouvroy's work in Belgium proves that satisfactory results can be obtained, with little outlay, whenever public cooperation and sympathetic interest are enlisted.

AMESSE, Denver.

Society Transactions

PEDIATRIC SECTION OF THE NEW YORK ACADEMY OF MEDICINE

Regular Meeting, Nov. 10, 1927

WILLIAM ROPES MAY, M.D., *Presiding*

XERODERMA PIGMENTOSA. DR. H. R. CRAIG.

Two cases of xeroderma pigmentosa were presented. The patients were sisters, aged 20 months and 5 years, respectively, and were of Italian parentage. The father, aged 41, and the mother, aged 31, were living and well. The eldest, 14 years of age, of a family of six children was living and normal. The second child died with xeroderma pigmentosa, with generalized carcinomatous metastases, at the age of 9 years. The third and fourth children were living and well. The fifth and sixth children were the patients.

CASE 1.—The patient, aged 5 years, was born in December, 1922, and four months later photophobia was first noticed, followed shortly by slight tanning and freckling. The child was first taken outdoors in June, at the age of 6 months. Feeding and developmental history were normal. The photophobia, freckling and general tanning became progressively more marked, especially on the exposed surfaces, until about a month before admission, when masses appeared on each lower eyelid. That under the right eye was a hard keratotic mass, irregular in contour though more or less pyramidal in general outline, grayish brown, avascular and entirely insensitive. On the lower left lid was a globular mass about 2 cm. in diameter, located near the inner canthus; it was elastic to the touch and telangiectatic, and it had some keratosis on its median surface. About its base there was some thickening and infiltration suggesting neoplasia probably of the acanthoma type. This mass was not tender. The skin over the entire body was markedly tanned, and there was abundant freckling on the exposed surfaces, with small miliary areas of telangiectasis and atrophy of the skin. In addition, several small areas of keratosis were present on the back of the neck. The mucous membranes were not involved; otherwise the physical examination did not reveal anything. The laboratory observations were as follows: hemoglobin, 60 per cent; red blood cells, 4,016,000; platelets, 464,000; white blood cells, 7,550; polymorphonuclears, 75 per cent, and lymphocytes, 25 per cent; fragility, normal; urine, normal; sugar tolerance, normal; blood sugar, 110 mg. per hundred cubic centimeters; blood nonprotein nitrogen, 40.5 mg. per hundred cubic centimeters; urea nitrogen, 15.7 mg. per hundred cubic centimeters; calcium, 11.2 mg. per hundred cubic centimeters; phosphorus, 5.0 mg. per hundred cubic centimeters; cholesterol, 0.121 mg. per cubic centimeter; van den Bergh, urine-urobilinogen and melanin tests, negative; carbon dioxide combining power, 55.5 per cent by volume.

CASE 2.—The feeding and developmental history of this patient, aged 20 months, was normal. Physical examination did not show any abnormalities, with the exception of marked generalized tanning over the entire body, with greatest pigmentation and freckling over the exposed surfaces, especially the face. Photophobia was marked. Keratoses or telangiectases were not present. The laboratory observations were similar to those in the older sister.

Two cases of xeroderma pigmentosa occurring in sisters, one in the earlier stages, the other presenting the later phases with skin atrophy, keratoses, telangiectases and carcinoma were presented.

NEUROBLASTOMA OF THE SUPRARENAL IN YOUNG CHILDREN. MARTHA WOLLSTEIN.

Primary tumors of the suprarenal medulla occurring in young children, congenital in origin and malignant in course, have been known for a long time. They are reported in the literature under various headings. In 1910, Wright studied this group of neoplasms and described them as neurocytoma or neuroblastoma of the suprarenal medulla. Wright drew attention to the similarity of the morphology and arrangement of the cells and fibrils in these growths to that in the anlage of the suprarenal and the sympathetic nervous system at certain periods of their development. On the basis of the most recent histologic investigation, Bailey and Cushing prefer the name sympathicoblastoma because the tumors originate from embryonal pluripotential cells of the sympathetic nervous system. While the majority of the reported tumors of this type have been found in or growing from the suprarenal medulla, they may grow from sympathicoblasts anywhere in the body. Thus tumors not involving the suprarenal gland have been reported by Landau from the abdominal sympathetic chain in a girl, aged 8 months, and by Boyd in a boy, aged 4 years and 6 months, while Martius described one from the cervical sympathetic system in a boy, aged 2 years and 6 months. In Pick's case the tumor grew from the uterus, and Anderson and Shennan found one growing in the apical region of the right lung in a girl, aged 12 weeks.

At the Babies' Hospital, a series of nine cases of suprarenal sympathicoblastomas, proved by microscopic study at operation or at autopsy, have accumulated. These nine neoplasms fall into several distinct groups clinically and anatomically; three occurred in boys and six in girls. The tumor involved the left suprarenal gland in four cases, the right in four cases and both glands in one instance. The age of the children varied from 3 weeks to almost 3 years, three being under the age of 1 year, three at the age of 2 and three, at the age of 3. There were five different types according to the location of the primary tumor and its metastases. Thus there is, first, the simplest case of the series, in which a small nodule was found in the left suprarenal, leaving the greater portion of the organ in an apparently normal condition, and forming no metastases. On microscopic examination, this tumor showed the most typical and exquisite structure of embryonal sympathetic cells and fibrils according to Wright's classic picture.

The next type was illustrated in the cases of two boys, aged 3 weeks and 9 months, respectively. The younger child showed the primary tumor in the right suprarenal; the elder, in the left. Both showed metastases in the liver enlarging it so greatly that this became the outstanding symptom. Other metastases were not present. While cases of this (Pepper) type occur most often in young infants, Morse and Wolbach have reported a case in a boy 4 years of age.

The third type conforms to what is known as the "Hutchison type of round cell sarcoma of the adrenal." It was represented in our series by two patients, one boy and one girl, aged 18 and 31 months, respectively. In the latter, the right suprarenal was affected, while in the former the left side was the seat of the primary growth. Both of these children showed metastases over a much wider area, involving bones of the skull, the ribs, the liver, and in the case of the girl, the dura mater as well. The liver was involved to a much less extent than was the case in the second group. The children thus affected are, in most cases, in the second or third year of life.

In the fourth group there were also two patients, a boy aged 23 months and a girl, aged 26 months. In the boy both suprarenals were affected; in the girl, only the left. Both were characterized by an inoperable tumor lying in front of the vertebral column between the suprarenals and kidneys, making a mass from which the kidneys, suprarenals and prevertebral neoplasms had to be painstakingly separated and dissected. The prevertebral mass was a second tumor focus, and a metastasis was present in one kidney.

The fifth group consisted of two cases in which the neoplasm was unattached to the kidney and to the surrounding structures and therefore operable. One of the children, a boy, aged 15 months, is alive one year after operation. The neoplasms from both these children showed areas of sympathicoblastoma and of ganglioneuroma; that is, in some parts of the growth the cells had become differentiated into ganglion cells, and these portions were firmer and whiter.

A case in which there was complete recovery has not been reported. The prognosis is practically always fatal, because either the growth is locally invasive or it metastasizes early, leaving only a small percentage of the cases operable when diagnosed.

THE EARLIER DIAGNOSIS OF MEASLES. PHILIP MOEN STIMSON.

As the coming half year will probably show a heavy incidence of measles, this disease apparently occurring in biennial waves in New York City, it would seem advisable to emphasize the familiar details in the early clinical diagnosis of measles, and, if possible, to suggest new or less well known features which might aid in the still earlier recognition of the presence of measles. Of the four periods of the disease, those of incubation, of invasion or catarrh, of the rash and of convalescence, only the first two are discussed here.

In the last half of the period of incubation there may be a temporary loss of from 5 to 6 ounces in weight, and the blood count may show a lymphocytic leukopenia toward the end of the period.

The period of invasion or catarrh in the great majority of cases begins with fever. Within a half day or so, a "measles line" or linear congestion may be found occasionally in the mucous membrane of the lower eyelid, about a third of the way from the blepharal margin to the fornix. In ten children in whom a typical measles line of this sort was observed in the eye and the disease prognosticated at this stage, it subsequently became manifest in eight. The duration of the line as such is short, the infection spreading to become a general conjunctivitis when the other evidences of catarrh appear, such as coryza and cough, comprising with conjunctivitis, the "three C's" characteristic of the second or third day of the disease and the cause of much of its spread to other people. The rash in the throat, or exanthem, usually precedes the "three C's," as may also a short-lived prodromal rash on the body. Koplik's spots inside the cheeks, which have also been reported as having been found in the eyes, nose and vagina, usually follow the catarrh in order of appearance; hence, the condition of patients in the stage of catarrh has been contagious for an appreciable length of time before the finding of Koplik's spots establishes the diagnosis. The typical exanthem characteristic of the disease follows usually at least a day later, after the appearance of the pathognomonic Koplik's spots.

Uncomplicated measles rarely kills, deaths being due almost without exception to the effects of secondary infections. Therefore every case of measles should be isolated at its onset from every other case. Experience in the hospital and at home has shown conclusively the value of this measure, and the harm that follows when it is not used.

Therefore, this winter and every year, physicians must be prepared to make the earliest possible diagnosis of measles, to isolate the patient on the first suspicion and to protect him from secondary infection.

DERMATOLOGIC CONDITIONS AS SEEN IN CHILDREN. A. BENSON CANNON.

Eczemas and impetiginous infections are the most frequently seen dermatologic conditions in infants and young children. For convenience, three groups of the commoner forms of eczemas are classified according to etiology as infantile, seborrheic and pyogenic.

Infantile eczema usually occurs in infants from 3 weeks to 6 months of age, appearing as a bilateral, symmetrical, diffuse, catarrhal dermatitis on the cheeks and often extending to other parts of the body. Seborrhea occurs

first on the scalp as a dry, finely scaly condition, and on spreading to the body, the lesions are sharply demarcated, erythematous patches, the scales being dry or slightly oily. Pyogenic eczema begins as staphylococcic infection beneath the vernix caseosa and gradually involves the scalp, face and body as a sharply defined red, crusted, purulent condition. Other conditions analogous to and simulating these eczemas but of different etiology and requiring somewhat different treatment are mycotic infections, infectious eczematoid dermatitis and so-called "candy" eczema.

Of the impetiginous infections, simple impetigo is the most prevalent, occurring usually on the exposed parts of the skin as an erythematous, vesicular, bullous, yellow and waxy, crusted condition. In new-born infants, the condition sometimes becomes generalized, appearing as an epidemic in hospital nurseries, and often ending fatally. Another impetiginous infection (Ritter's disease) also occurs in the new-born as a generalized, moist, exfoliative dermatitis of streptococcic origin. This type also frequently ends in death. There is a follicular impetigo (Bockhardt), usually beginning as papular and pustular eruption, which often spreads over the entire body and extremities, and which may prove fatal.

Two dermatoses which are encountered in early life are characterized by intense itching and prurigomitis and chronic lichen simplex. While dissimilar, both have often been attributed to protein sensitization, and both are chronic.

The ability to distinguish between the several types of eczemas and to recognize the form of treatment indicated often enables one to predict with some degree of certainty the results which can be obtained in the individual case. While it is impossible to effect a cure in all children suffering from true infantile eczema, the condition can always be alleviated for a longer or shorter period. Impetiginous infections, can usually be promptly and permanently cured by the ordinary antiseptic and antiparasitic remedies; sometimes this cannot be done in the new-born.

ST. LOUIS PEDIATRIC SOCIETY

Regular Meeting, Nov. 4, 1927

PARK J. WHITE, M.D., *in the Chair*

TREATMENT OF ERYSIPELAS WITH MERCUROCHROME-220 SOLUBLE AND POLYVALENT ANTISTREPTOCOCCUS SERUM. DR. GEORGE GARRISON.

Twenty-nine patients with erysipelas of severe degree were selected from approximately 100 patients with this disease admitted consecutively to the Isolation Hospital of St. Louis. Seventeen were given a commercial polyvalent antistreptococcic serum intravenously or intramuscularly in large doses. Eleven patients of this group recovered, and eight of them showed a critical drop in temperature and a rapid improvement in the local and general condition. It was found that from 50 to 100 cc. of this serum was necessary to produce a definite effect on the course of the disease. Usually about 50 cc. was required for children and 100 cc. for adults. Unfavorable results were not observed except that most of the patients developed urticaria after a few days.

Twelve patients were given mercurochrome-220 soluble intravenously in 1 per cent solution, the dose for adults being from 15 to 25 cc., which was repeated in twelve hours if necessary. Only five patients received two injections and none received a total of more than 40 cc. Nine patients in this group recovered, and seven of them showed an abrupt drop in temperature and a striking improvement in the general and local condition. Complications or sequelae resulting from the use of mercurochrome-220 soluble were not observed.

All of the 100 patients, those with mild and severe cases alike, were treated locally with cold saturated solution of magnesium sulphate, and this may be disregarded as a factor in considering the course of these special groups.

The temperature reaction, the toxic symptoms, the local manifestations and the entire course of the disease were definitely cut short in the patients who responded to these two methods of treatment.

Though the percentage showing definite and immediate improvement was approximately the same in the two groups, those who received the mercurochrome showed a much more striking effect.

A Case of Congenital Hemolytic Icterus. Dr. T. S. Zahorsky.

A boy, the fourth child of a family, none of whom had had jaundice, developed intense icterus on his second day. He slowly weakened, and the jaundice deepened during the next few days. He had frequent thin, yellow stools; the urine contained bile, and his spleen was large and firm. He never had fever. On the ninth day, the red cell count was 2,050,000, and the hemoglobin was 42 per cent. After a transfusion of 90 cc. of citrated blood, he improved for two days, but then the jaundice became worse and the red cell count fell again to 2,000,000 on the fifteenth day. Another similar transfusion was followed by steady improvement. On the twenty-first day, the jaundice had practically disappeared. The red cells totalled 3,500,000, and the hemoglobin was 61 per cent. A few nucleated red blood cells were seen, and 10 per cent of the erythrocytes were reticulated. There was a slight increase in fragility, complete hemoglobin occurring at 0.34 per cent. When the infant was 10 weeks old, the spleen remained large, but he appeared normal otherwise.

A Case of Agranulocytic Sepsis. Dr. John Zahorsky.

A child, aged 6 years, a mongolian idiot, had always been pale. Two weeks before the fatal outcome, he had a tooth extracted, after which the glands in the neck became enlarged and a purulent discharge appeared in the nares. A double otitis media was treated by puncture of the membranes of the drum. Petechial spots appeared on the body. The leukocyte count was 1,000, with only 2 per cent of granulocytes before death.

A Case of Scrofula Mistaken for Swift's Disease. Dr. John Zahorsky.

The infant at first showed photophobia, extreme restlessness, anorexia, loss in weight, excessive perspiration and pink hands and feet. The leukocyte count was 14,000.

The general symptoms suggested Swift's disease, but subsequently, a positive Pirquet test, the appearance of swollen glands in the neck, mucopurulent discharge from the nose and a marked conjunctivitis suggested tuberculosis. The glands in the neck broke down, were opened and left a large necrotic area in the skin which was successfully treated by means of the quartz light.

A Case of Giardiasis. Dr. John Zahorsky.

A girl, aged 10 years, had been suffering from repeated attacks of abdominal pain, sometimes so severe as to cause her to remain in bed for half a day. The bowels showed a tendency to looseness. The attacks had been diagnosed as appendicular colic. Examination of the stools showed an enormous number of giardial cysts. She was given bismuth salicylate, and the symptoms disappeared, at least temporarily.

A Case of Urticaria Pigmentosa. Dr. John Zahorsky.

In an infant boy, aged 4 months, who was breast-fed, papular pigmented lesions appeared when the child was 2 months old and were scattered over the trunk, scalp and extremities. Cod liver oil and orange juice had been given in addition to breast milk. The papules seemed to itch greatly at times. The general health was not affected. Intensive treatment with quartz light did not have any effect on the lesions. After four months' observation, the eruptions seemed about the same.

Book Reviews

KEEPING THE BABY WELL. By JOHN HOWELL WEST, PH.B., M.D. Price, $2. Pp. 227. New York: The Knickerbocker Press, 1927.

In this book the author discusses the care of children up to the school age, but special emphasis is placed on the first two years of life. The book is written for the mother in order to give her information about the routine care of her baby. The advice contained in the book is, on the whole, sound. In some places the mother is likely to be confused by a pseudoscientific discussion of the facts on which the advice is based.

The advice for feeding corresponds in general with modern practice. The recommendation for three hour intervals for bottle babies is somewhat more conservative than is generally in vogue at the present time. In fact, the whole tendency of the book is conservative, which perhaps is desirable considering that it is for lay use.

The occasional references to work at the Easton Hospital gives local flavor which detracts rather than adds to the general value. There are some matters which might be selected for adverse criticism, such as the use of a swab on a match stick for cleaning the baby's nose, and the casual remark that if a purulent discharge from the eye in a new-born baby does not clear up in a day or so, a physician should be consulted. There is some confusion, especially in the chapter on colic between conditions in infants and those in older children.

The recipes for preparing the baby's foods at the back of the book will be useful.

TOBACCO AND PHYSICAL EFFICIENCY. PIERRE SCHRUMPF-PIERRON, M.D., with a preface by PROF. HENRI VAQUEZ. Price, $1.85. Pp. 134. New York: Paul B. Hoeber, Inc., 1927.

In this book published under the Auspices of the Committee to Study the Tobacco Problem, the author has gone into the question offering an unbiased view of the material at hand. Seven hundred and fifty bibliographic references are given covering seventy pages. From these the author concludes that a sound individual may bear, what is for him, moderate doses without injury, but even these are often noxious to the unsound or other sound individuals. However, the immoderate use of tobacco brings on a series of disturbances which are first functional, then organic, and some are not without gravity.

Directory of Pediatric Societies

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SECTION OF DISEASES OF CHILDREN

Chairman: Dr. Hugh McCulloch, 500 S. Kingshighway, St. Louis.
Secretary: Dr. C. A. Aldrich, 545 Lincoln Ave., Winnetka, Ill.
Place: Minneapolis. Time: June 11-15, 1928.

AMERICAN PEDIATRIC SOCIETY

President: Dr. J. Claxton Gittings, 1828 Pine St., Philadelphia.
Secretary: Dr. Howard Childs Carpenter, 1805 Spruce St., Philadelphia.
Place: Washington, D. C. Time: April 30 to May 2, 1928.

THE ASSOCIATION OF AMERICAN TEACHERS OF THE DISEASES OF CHILDREN

President: Dr. J. P. Crozier Griffith, 1810 Spruce St., Philadelphia.
Secretary: Dr. Paul W. Emerson, 86 Bay State Road, Boston.
Place: Minneapolis. Time: June 12, 1928.

AMERICAN CHILD HEALTH ASSOCIATION

President: Hon. Herbert Hoover, 370 Seventh Ave., New York.
Secretary: Dr. Philip Van Ingen, 370 Seventh Ave., New York.
Place: Undecided. Time: May or June, 1928.

CANADIAN SOCIETY FOR THE STUDY OF DISEASES OF CHILDREN

President: Dr. G. R. Pirie, 20 Madison Ave., Toronto.
Secretary: Dr. A. P. Hart, 1844 Bloor St. West, Toronto.
Place: Vancouver, B. C. Time: June, 1928.

SECTIONAL

CENTRAL STATES PEDIATRIC SOCIETY

President: Dr. H. T. Price, 121 University Place, Pittsburgh.
Secretary: Dr. A. H. Parmelee, 310 S. Michigan Ave., Chicago.

SECTION ON PEDIATRICS OF SOUTHERN MEDICAL ASSOCIATION

President: Dr. Robert A. Strong, Pass Christian, Miss.
Secretary: Dr. Hugh L. Dwyer, Medical Arts Bldg., Kansas City, Mo.
Place: Memphis, Tenn. Time: Nov. 14 to 17, 1927.

LOCAL

NORTHERN CALIFORNIA PEDIATRIC SOCIETY

President: Dr. W. L. Channell, Medical Bldg., Oakland, Calif.
Secretary: J. E. Parrott, Medico-Dental Bldg., San Francisco.
Place: Various Teaching Schools. Time: Monthly.

SOUTHWESTERN PEDIATRIC SOCIETY

President: Dr. Oscar Reiss, Westlake Professional Bldg., Los Angeles.
Secretary: Dr. William Sidney Bowers, 1136 West 6th St., Los Angeles.
Place: University Club of Los Angeles. Time: First Wednesday in January, March, May, September and November.

THE ROCKY MOUNTAIN PEDIATRIC SOCIETY

President: Dr. W. Wiley Jones, Majestic Bldg., Denver.
Secretary: Dr. Berryman Green, 537 Republic Bldg., Denver.
Place: Children's Hospital, Denver. Time: Third Saturday of each month, 2:30 p. m., September to May, inclusive.

PEDIATRIC SECTION OF FULTON COUNTY MEDICAL SOCIETY, ATLANTA, GA.

President: Dr. William W. Anderson, 362 Peachtree St., Atlanta.
Secretary: Dr. Lee Bivings, Exchange Bldg., Atlanta.
Place: Academy of Medicine, 32 Howard St., Atlanta.
Time: Second Thursday of each month from October to April, at 8 p. m.

NEW ENGLAND PEDIATRIC SOCIETY

President: Dr. James Herbert Young, 66 Commonwealth Ave., Boston.
Secretary: Dr. Thomas Lanman, 286 Marlborough St., Boston.
Place: Boston Medical Library. Time: Second Friday of each month.

CHICAGO PEDIATRIC SOCIETY

President: Dr. Maurice Blatt, 30 N. Michigan Ave., Chicago.
Secretary: Dr. J. K. Calvin, 104 S. Michigan Ave., Chicago.
Place: City Club. Time: Third Tuesday of each month.

LOUISIANA STATE PEDIATRIC SOCIETY

President: Dr. L. R. De Buys, 1487 Delachaise St., New Orleans.
Secretary: Dr. F. J. Kinberger, 3601 Prytania St., New Orleans.

DETROIT PEDIATRIC SOCIETY

President: Dr. George E. White, Capitol Theatre Bldg., Windsor, Ontario, Canada.
Secretary: Dr. W. S. O'Donnell, 1563 David Whitney Bldg., Detroit.
Place: Children's Hospital of Michigan. Time: No fixed date; approximately once a month.

NORTHWESTERN PEDIATRIC SOCIETY

President: Dr. F. W. Schlutz, University of Minnesota, Minneapolis.
Secretary: Dr. E. S. Platou, 730 La Salle Bldg., Minneapolis.
Place: University Campus and Rochester. Time: Winter, Spring, Summer, Fall.

ST. LOUIS PEDIATRIC SOCIETY

President: Dr. T. Wistor White, Lister Bldg., St. Louis.
Secretary: Dr. E. H. Rohlfing, 816 Beaumont Medical Bldg., St. Louis.
Place: St. Louis Children's Hospital. Time: At least once a year and contingent thereafter on the arrangement of a suitable program.

NEBRASKA-IOWA PEDIATRIC SOCIETY

President: Dr. Floyd Clarke, 731 City Nat'l Bank Bldg., Omaha.
Secretary: Dr. R. B. Eldredge, 1036 Medical Arts Bldg., Omaha.
Place: Add-Sell Restaurant. Time: Second Thursday of each month from October to May, inclusive, 6 p. m.

BROOKLYN PEDIATRIC SOCIETY

President: Dr. Harold L. Barnes, 1235 Dean St., Brooklyn.
Secretary: Dr. Charles A. Weymuller, 61 Greene Ave., Brooklyn.
Place: Kings County Medical Society Bldg. Time: Last Wednesday of each month, except June, July and August.

BRONX PEDIATRIC SOCIETY

President: Dr. William Hinz, 1964 Grand Concourse, New York.
Secretary: L. H. Barenberg, 1749 Grand Concourse, New York.
Place: Concourse Plaza, 161st, St., and Grand Concourse, New York.
Time: Second Wednesday of each month, except June, July, August and September.

CENTRAL NEW YORK PEDIATRIC CLUB

President: Dr. Frank Vander Bogert, 111 Union St., Schenectady, N. Y.
Secretary: Dr. Frank J. Williams, 58 S. Swan St., Albany, N. Y.
Place: Various Cities in New York. Time: Fourth Thursday of October, January and April.

PEDIATRIC SECTION OF THE NEW YORK ACADEMY OF MEDICINE

President: Dr. William Ropes May, 8 East 66th St., New York.
Secretary: Dr. Stafford McLean, 17 East 71st St., New York.
Place: New York Academy of Medicine, 2 East 103d St. Time: Second Thursday of each month from October to May, inclusive, 8:30 p. m.

THE PEDIATRIC SECTION OF THE ACADEMY OF MEDICINE OF CLEVELAND

Chairman: Dr. J. W. Epstein, 3912 Prospect Ave., Cleveland.
Secretary: Dr. Charles W. Burhans, 14805 Detroit Ave., Lakewood, O.
Place: 2009 Adelbert Road. Time: Four meetings, from September to May.

PEDIATRIC SECTION, ACADEMY OF MEDICINE, TORONTO

President: Dr. M. A. Cox, 34 St. Clair Ave. West, Toronto.
Secretary: Dr. F. R. Hassard, 1361 King St. West, Toronto.
Time: November 17, December 15, February 16 and March 15.

PHILADELPHIA PEDIATRIC SOCIETY

President: Dr. Edward L. Bauer, Jefferson Medical College, Philadelphia.
Secretary: Dr. John Porter Scott, 2040 Pine St., Philadelphia.
Place: College of Physicians, 19 S. 22nd St., Philadelphia. Time: Second Tuesday of each month from October to June, inclusive.

MEMPHIS PEDIATRIC SOCIETY

President: Dr. W. D. Blue, 1024 Madison Ave., Memphis, Tenn.
Secretary: Dr. Gilbert J. Levy, 899 Madison Ave., Memphis, Tenn.
Place: Memphis General Hospital. Time: Second Tuesday of each month.

TEXAS PEDIATRIC SOCIETY

President: Dr. Lucius D. Hill, Medical Arts Bldg., San Antonio.
Secretary and Treasurer: Dr. Edwin G. Schwarz, Medical Arts Bldg., Fort Worth.
Place: Houston. Time: December, 1927.

RICHMOND PEDIATRIC SOCIETY

President: Dr. Howard Urbach, 1331 Grove Ave., Richmond, Va.
Secretary: Dr. W. Ambrose McGee, West End Medical Bldg., 2618 Grove Ave., Richmond, Va.
Place: Westmoreland Club, 601 E. Grace St., Richmond, Va.
Time: First Thursday of each month (other than during the summer).

VIRGINIA PEDIATRIC SOCIETY

President: Dr. Lawrence T. Royster, University, Va.
Secretary: Dr. William B. McIlwaine, Petersburg, Pa.
Place: Danville, Va. Time: October 17, 1928.

NORTH PACIFIC PEDIATRIC SOCIETY

President: Dr. R. S. Miles, Fidelity Bldg., Tacoma, Wash.
Secretary: Dr. D. M. Dayton, 704 St. Helens Ave., Tacoma, Wash.
Place: Seattle. Time: January, 1928.

SEATTLE PEDIATRIC SOCIETY

President: Dr. George B. McCulloch, Cobb Bldg., Seattle.
Secretary: Dr. C. L. Smith, Stimson Bldg., Seattle.
Place: College Club. Time: Third Friday of each month, at 7 p. m.

MILWAUKEE PEDIATRIC SOCIETY

President: Dr. Roy M. Greenthal, 141 East Wisconsin Ave., Milwaukee.
Secretary: Dr. M. G. Peterman, 167 Seventeenth St., Milwaukee.
Place: Children's Hospital, Milwaukee. Time: Second Wednesday of each alternate month, beginning with February.